CLINICAL PROFILE OF ARRHYTHMIAS IN GOLDEN HOUR OF MYOCARDIAL INFARCTION

Dissertation Submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulations for the award of the degree of

MD GENERAL MEDICINE

BRANCH- 1

TIRUNELVELI MEDICAL COLLEGE

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY CHENNAI INDIA

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PROF . DR. GEETHA RANI. MD
PROFESSOR AND HEAD
DEPARTMENT OF MEDICINE
TIRUNELVELI MEDICAL COLLEGE HOSPITAL
TIRUNELVELI.

DR.M.MANOHARAN. MS
DEAN
TIRUNELVELI MEDICAL COLLEGE HOSPITAL
TIRUNELVELI

DECLARATION

DR.C.ANANTHI, solemnly declare that this dissertation **"CLINICAL PROFILE OF ARRHYTHMIAS IN GOLDEN HOUR OF MYOCARDIAL INFARCTION"** is a bonafide record of work done in DEPARTMENT OF MEDICINE, GOVERNMENT TIRUNELVELI MEDICAL COLLEGE & HOSPITAL, TIRUNELVELI under the guidance of **PROF.DR.GEETHARANI MD.,** GOVERNMENT TIRUNELVELI MEDICAL COLLEGE HOSPITAL, TIRUNELVELI.

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CONTENTS

ABBREVIATIONS

SA Node – Sino atrial node

AV Node-Atrio Ventricular Node

ICCU- Intensive Coronary Care Unit

AV Block- Atrioventricular Block

LAHB - Left Anterior Hemiblock

LPHB – Left Posterior Hemiblock

LBBB – Left Bundle BranchBlock

RBBB –Right Bundle Branch Block

LAD – Left Anterior Descending Artery

LCX – Left Circumflex Artery

S 1 – 1 Septal branch

D 1 – 1 Diagonal branch

SHT – Systemic Hypertension

DM – Diabetes Mellitus

INTRODUCTION

Acute myocardial infarction is the commonest cause of death and disability and it poses great economic burden than any other ailments. In AMI the main cause for mortality is arrhythmias, which are due to a autonomic nervous system imbalance, electrolyte imbalance and ischemia which causes conduction blockade in the infarcted zone.

Even though there are vast advances in the mode of diagnosis and management of Infarction related arrhythmias over the past 25 years it continues to be a major cause of mortality in industrialized world and is emerging as a great health hazard in developing countries.The development of acute myocardial infarction is a fatal event in about one third of the patients, and among this,half of the deaths occurs due to ventricular tachyarrhythmias that occurs within one hour of the event.

So to reduce the incidence of mortality careful monitoring of cardiac rhythm and accurate management of arrhythmias in the golden hour of infarction is essential.

Several phases in the management of patients lead to decline in mortality from Acute myocardial infarction."CLINICAL

OBSERVATION PHASE " – Focussed on detailed recording of physical & laboratory findings, treatment consists of strict bed rest and sedation.

"CORONARY CARE UNIT PHASE " – Focussed on detailed analysis and vigorous management of arrhythmias.

"HIGH TECHNOLOGY PHASE" - Which is done by the introduction of interventions like pulmonary artery floatation catheter for bedside monitoring of haemodynamics and more precise management of heart failure.

Lawn and his colleagues, by their proactive works found that prevention of ventricular arrhythmias related death in AMI could be achieved only through intensive coronary care.

During the past 25 years the mortality has been reduced from 30% to about 15% due to the vast advancement in the Intensive coronary care and timely diagnosis and prompt management of serious life threatening arrhythmias.

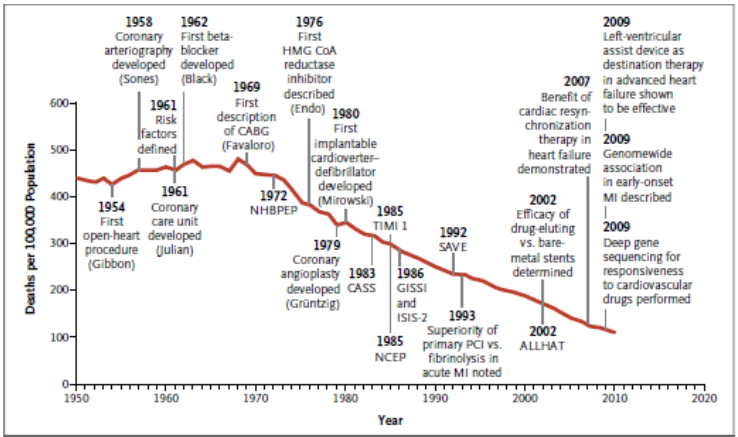


Figure 1. Decline in Deaths from Cardiovascular Disease in Relation to Scientific Advances.

The timeline shows the steady decline in cardiovascular deaths over the late 20th and early 21st centuries, along with major advances in cardiovascular science and medicine. ALLHAT denotes Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial, CASS Coronary Artery Surgery Study, GISSI Italian Group for the Study of Streptokinase in Myocardial Infarction, HMG-CoA 1-hydroxy-3-methylglutaryl coenzyme A, ISIS-2 Second International Study of Infarct Survival, MI myocardial infarction, NCEP National Cholesterol Education Program, NHBPEP National High Blood Pressure Education Program, PCI percutaneous coronary intervention, SAVE Survival and Ventricular Enlargement, and TIMI 1 Thrombolysis in Myocardial Infarction 1.

This is achieved in the Intensive coronary care units by the continuous monitoring of cardiac rhythm of each patient and hemodynamic monitoring in selected patients. Defibrillators, Respirators, Pacemakers, Pacingcatheter s and also trained staffs who can recognize arrhythmias and administer antiarrhythmics and perform cardiac resuscitation if necessary.

.Arrhythmias affect the prognosis of the patient after AMI by causing hemodynamic instability and also by directly affecting the myocardial contractility. The common type of arrhythmias in anterior wall myocardial infarction is tachyarrhythmias while in inferior wall myocardial infarction bradyarrhythmias are common.

Timely restoration of flow in the epicardial infarct related artery combined with improvement of the downstream zone of the infracted myocardium ultimately results in a limitation of infarct size.Because the recognition and treatment of arrhythmias in the first hour not only reduces the in- hospital mortality , but also the long term survival is also excellent in those patients who survive after primary ventricular fibrillation (VF that occurs in early hours and is not associated with any

predisposing factors like CHF, shock, bundle branch block or ventricular aneurysm)

Various studies have analysed the effect of different types of arrhythmias in a individually but data from India is insuficient. In this study we are going to analyse the incidence and prognosis of various types of arrhythmias in the "Golden hour of myocardial infarction".

The "Golden hour of myocardial infarction" is the first 60 minutes . "Total ischaemic time" is within 120 minutes. The first 60 minutes of myocardial infarction is important because during an attack of acute myocardial infarction while the central zone of the infarct contains the necrotic tissue that is irreversibly lost , the fate of surrounding ischaemic myocardium (ischaemic penumbra) may be improved by timely restoration of coronary perfusion , reduction of myocardial O_2 demands , prevention of the accumulation of noxious metabolites , and blunting the impact of mediators of reperfusion injury (eg. Calcium overload and oxygen derived free radicals)

AIM OF THE STUDY

The aim of the study is to analyse:

1. The incidence of different types of arrhythmias in 100 patients with acute MI admitted to intensive coronary care unit of Tirunelveli Medical College Hospital.

2. Correlation of each type of arrhythmia with respect to location and extension of acute myocardial infarction.

3. To assess the risk factors like age, sex , personal habits in relation to the various types of arrhythmias.

4. To assess the prognostic factors in various types of arrhythmias and their outcome.

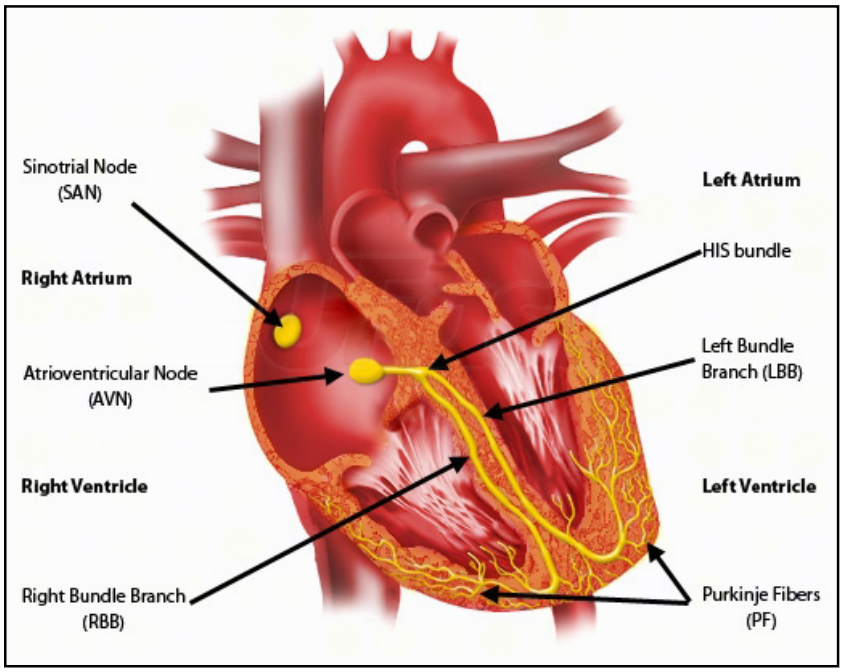
5. To interpret effect of arrhythmias on the mortality and morbidity in patients in the golden hour acute myocardial infarction.

REVIEW OF LITERATURE

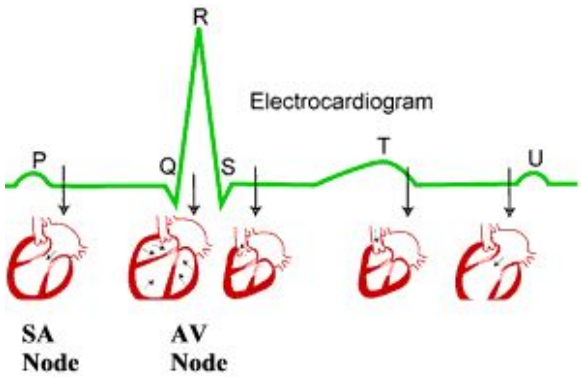
HISTORICAL ASPECTS

The field of electrophysiology was ushered in the Twentieth century- As the Ducth physician Einthoven introduced Electrocardiography. In 1960- Development of intracavitary recording, particularly, His bundle electrogram with programmed stimulation of heart , marked the beginning of contemporary clinical electrophysiology. In 1990s-Interventional cardiac electro physiology came in to existence after the adoption of radiofrequency technology to ablate cardiac tissue.[1]

Anatomy and Physiology of the conduction system:

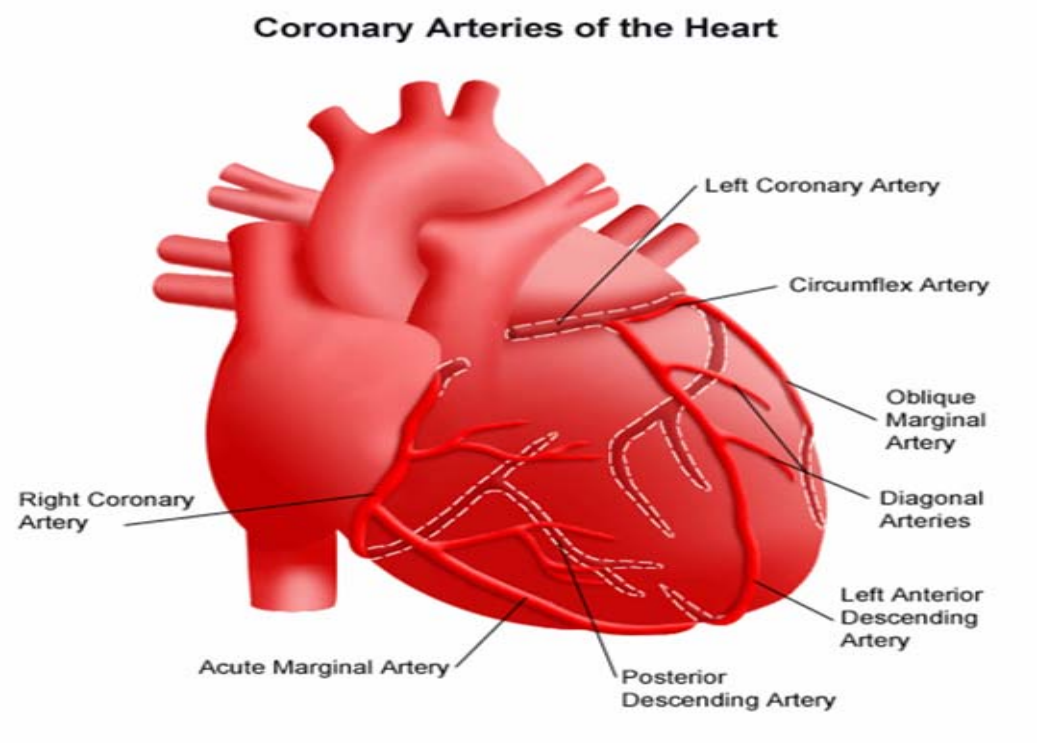


The sinoatrial node (SANode) is the pacemaker of the heart where the normal cardiac impulses are generated, which is located at the juction of the right atrium and superior vena cava.[2] This impulse is slowly transmitted through the nodal tissue to the anatomically complex atria, where it is more rapidly conducted to the atrioventricular node (AVN) inscribing the `p' wave of the ECG.



There is a perceptible delay in conduction through the anatomically&functionally heterogenous AVN. The time needed for activation of the atria and the AVN delay is represented as the PR interval of the ECG. The impulses travel through the right & left Bundle of His and through the purkinje fibres and ventricles are activated inscribing a QRS complex .[4]Recovery electrical excitability results in repolarisation occurring first on the epicardial surface then proceeding to the endocardium which inscribes a T wave normally.

BLOOD SUPPLY OF HEART & CONDUCTION SYSTEM



The arterial blood supply of the heart is derived from two coronary arteries – Right coronary & Left coronary arteries . Left coronary artery arises from left sinus of valsalva and it divides in to left anterior descending and left circumflex arteries.

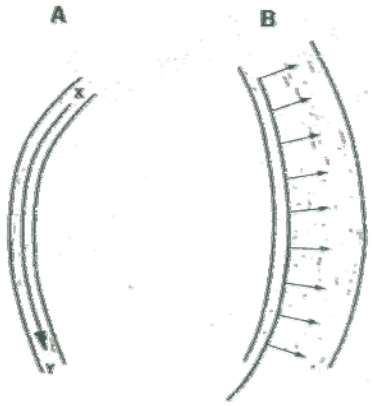
Left anterior descending artery supplies the interventricular septum and anterolateral wall of the left atrium . Left circumflex supplies the left atrium and lateral wall of left ventricle.

Right coronary artery arises from the Right sinus of valsalva and it supplies right atrium , right ventricle , posterior part of interventricular septum and inferior part of left ventricle.The subendocardial region

directly receives its blood supply from blood in the ventricular cavity.The least perfused zone of myocardium is subendocardial region and so it is most susceptible to ischaemia.

SA node & AV node are supplied by Right coronary artery.The right bundle branch and the posterior division of left bundle have a dual blood supply from the left anterior descending and right coronary arteries, whereas the anterior division of left bundle branch is supplied by septal perforators originating from the left anterior descending coronary artery.

ATRIAL & VENTRICULAR ACTIVATION- The biatrial chamber is a relatively thin walled structure and is not equipped with the highly specialized conducting system like the ventricles.[6] Activation of the biatrial chamber therefore occurs longitudinally and by contiguity, spreading from its point of origin in the SA node to engulf the whole chamber, each fibre inturn activating the adjacent fibre.



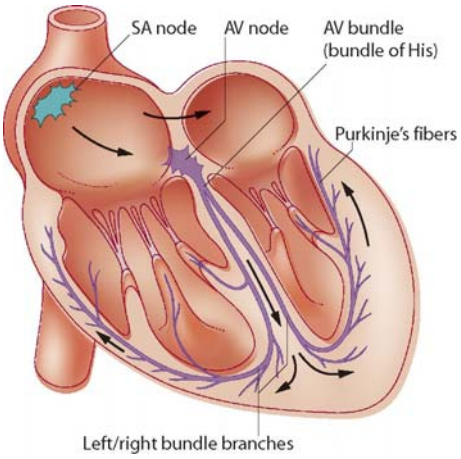
(A) Atrial activation (B) Ventricular activation

Activation of the ventricles is effected through the specialized and highly efficient conduction system which transmits the supraventricular impulses very rapidly to all the endocardial regions of the chamber. The muscle is then activated from endocardial to epicardial surfaces through the terminal ramifications of the conducting system.[12] Excitation therefore occurs transversely through the ventricular myocardium and this enables the whole chamber to be activated near synchronously.

The pacemakers of the heart:

There are many pacemakers in the heart. Pacemaker cells are situated in the SA node which is situated in the right atrium, the AV node the only electrical connection between the atria and the ventricles, the bundle of His which divides in to right and left bundle, the atria and the ventricles (every purkinje cell itself is a potential pacemaking cell). The

rate of SA node is 70–80 beats/minute. The rate of AV nodal pacemaking cells is 60 beats/mt. In the bundle of His is 50 beats/mt. The rate of punkinje cells of the ventricular muscle is 30 to 40 beats/mt. From this it is evident that if the potential pacemaker is away from the SA node the inherent rate of discharge will be slower.[13]



Thus it is evident that there is not only one pacemaker governing the heart rate, in situations if the fastest pacemaker defaults the slower pacemaker takes over the pacemaking function . AMI may be associated with SA node dysfunction (especially in inferior wall MI) but the abnormalities are transient.

Mechanism of cardiac arrhythmias:

Cardiac arrhythmias result from abnormalities of electrical impulse generation, conduction or both.

Bradyarrhythmias typically arise from[15]

1. Disturbances in impulse formation at the level of the SA node.
2. Disturbances in impulse propagation at any level including exit block from the SA node, conduction block at the AV node, impaired conduction in the His punkinje system.

Tachyarrhythmias : *due to*

1. Enhanced automaticity (spontaneous depolarization of atrial, junctional or ventricular pacemaker)
2. Reentry (circus propagation of a depolarization wave from)
3. Triggered arrhythmias (initiated by after depolarization).

Classification of arrhythmias:

Bradyarrhythmias:[16]

1. Sino Atrial block
2. Intra atrial conduction block
3. AtrioVentricular dissociation
4. AtrioVentricular blocks

First – degree AV block (1^0)

Second – degree AV block(2^0)

Mobitz typeI (wenckebech)

Mobitz type II

Third degree or complete heart block (3^0)

Tachyarrhythmias:[18]

Supraventricular tachyarrhythmias

1. Sinus tachycardia
2. Atrial premature complexes
3. Atrial fibrillation
4. Junctional premature complexes
5. Atrial flutter and atrial tachycardia
6. AVRT / AVNRT

Ventricular tachyarrhythmias

1. Ventricular premature complexes
2. Ventricular tachycardia.
3. Accelerated idioventricular rhythm

Arrhythmias in myocardial infarction:

The incidence of arrhythmias is higher in patients the earlier they are seen after the onset of symptoms. Many serious arrhythmias develop before hospitalization. Almost 90% of the patient develop rhythm abnormalities after acute myocardial infarction, 67% of ventricular arrhythmias occur within 12 hours of MI, 25% of patients experience conduction disturbances within 24 hours.

Arrhythmic Complications of MI:[19]

Now a days with widespread use of fibrinolytics and primary interventions, arrhythmias are observed less frequency. But arrhythmias are more common in patients with the following conditions.

- ❖ Anterior MI
- ❖ Infarct involving a larger area
- ❖ MI complicated by congestive heart failure
- ❖ MI associated with hypotension and hypoperfusion
- ❖ MI in older patients
- ❖ Re infarction

The prompt management of arrhythmias constitutes a significant advance in the treatment of MI.

Tachyarrhythmias during acute MI are due to

- ❖ Abnormalities of impulse formation.
- ❖ Abnormalities in impulse propagation due to ischaemia of the infarcted zone.

Bradyarrhythmias,[21] during the initial few hours of acute myocardial infarction, usually results from enhanced vagal activity, which is usually benign. But conduction disturbances beyond first 24 hours requires most attention. It indicates necrotic damage of the

conduction tissue, these conduction blocks will produce much complications.

There are emerging clinical techniques to measure the area at risk for arrhythmias. Measurement of certain parameters with MRI (surface area of the infarct and amount of ventricular mass damaged by the infarct) predict the incidence VT better than EF measured by echocardiogram. The extent of periinfarct zone measured by MRI is correlated with risk of arrhythmias.[23]

Important arrhythmias in acute myocardial infarction:

Tachyarrhythmias:[24]

1. **Supraventricular arrhythmias**
   - ❖ Sinus tachycardia
   - ❖ Atrial fibrillation
   - ❖ Atrial flutter
2. **Ventricular**
   - ❖ Ventricular premature beats
   - ❖ Ventricular tachycardia and fibrillation
   - ❖ Accelerated idioventricular rhythm

Brady arrhythmias:[26]

- ❖ Sinusbradycardia
- ❖ Atrioventricular and intraventricular conduction disturbances.

Supra ventricular arrhythmias:

Sinus tachycardia:[30]

Sinus tachycardia is the most common supra ventricular arrhythmia in patients with acute myocardial infarction. It occurs mainly due to symphathetic over stimulation (e.g. as a part of hyperdynamic state).

It may occur secondary to other causes like fever, hypotension, hypovolemia, heart failure, drugs (nicotine, caffeine).

Treatment:

If it occurs secondary to another causes as mentioned above the primary problem should be treated first.

If it occurs due to sympathetic over stimulation then treatment with a beta blocker is indicated. calcium channel blockers (verapamil and diltiazem) can also be used. If it is resistant to drug therapy sinus node radiofrequency modification or surgical ablation can be done.

Atrial fibrillation:[32]

Atrial fibrillation is also a most common type of arrhythmia after acute myocardial infarction which are often secondary to left ventricular failure, infarction in the atria or right ventricular myocardial infarction.

The incidence of atrial fibrillation increases with age, and in patients with associated risk factors like systemic HT, Ischemic heart diseases, Diabetes mellitus.

Atrial fibrillation also occurs in patients of acute MI with high adrenergic drive, associated hypoxia, hypokalemia, hypoglycemia.

AF has clinical importance related to

1. The loss of atrial contractility
2. The inappropriate fast ventricular response and
3. The loss of atrial appendage contractility and emptying leading to the risk of clot formation and subsequent thromboembolic events.

Treatment:

Acute treatment of atrial fibrillation that is symptomatic with an increased ventricular rate > 120 bpm should include consideration of urgent synchronized cardioversion (100 – 200 J monophasic wave form) if the patient is haemodynamically unstable. (e.g. hypotensive) or has evidence of ischaemia or pulmonary edema.[38]

Digoxin is usually the treatment of choice for supra ventricular arrhythmias it heart failure is present.

If heart failure is absent beta blockers, verapamil or Diltiazem are suitable alternatives for controlling the ventricular rate, as they may also help to control ischemia.

Rhythm control:

A single episode of AF does not require any intervention or a short course of beta blocker therapy can be given.

In cases of recurrent AF with significant structural heart disease the antiarrhythmics sotalol, amiodarane, dofetilide or dronedarane can be used.

In patients without evidence of structural heart disease or hypertensive heart disease, the use of class IC antiarrhythmic agents, flecainide or propafenone can be used.[39]

Atrial flutter:

Atrial flutter after acute myocardial infarction occurs in < 5% of patients is also due to ischaemia of the infarcted zone and LV failure more commonly occurs in atrial infarctions especially of right atrium. The organized atrial flutter activity frequently can be terminated with low energy DC cardioversion.

However if atrial flutter, does occur and is accompanied by symptoms, long term suppressive therapy is indicated. In all patients, an effort should be made to control the ventricular rate pharmacologically or restore sinus rhythm. Rate control with calcium channel antagonists

(Diltiazem or verapamil), beta blockers, and / or digoxin may be difficult.[39]

In selected patients with high anesthetic risk an attempt at pharmacologic cardioversion with procainamide, amiodarone or ibutilide is appropriate. Patients who manifest with recurrent AFL appear to be effectively treated with catheter ablative therapy. However its role in patients with a recent myocardial infarction have not been evaluated.

Ventricular arrhythmias:

Ventricular tachyarrhythmias after acute myocardial infarction are

- premature ventricular complexes.
- Non sustained or sustained VT
- Ventricular fibrillation and
- Polymorphic VT

Coronary reperfusion during thrombolysis or PCI has been associated with accelerated idioventricular rhythm and ventricular tachyarrhythmias. Non sustained or sustained VT often results from reentry and can occur late after myocardial infarction.

Ventricular premature beats:

Prior to the widespread use of reperfusion therapy, aspirin, beta blockers and intravenous nitrates in the treatment of acute MI, frequent

ventricular premature beats (>5 / min), ventricular premature complexes with multiform configuration, early coupling (the R-on-T phenomenon) can lead to ventricular fibrillation.[40]

Now it is clear that such warning arrhythmias don't have any clinical significance Primary VF can occur without any antecedent warning arrhythmias and also occurs even after suppression of warning arrhythmias.

Treatment:

Anti arrhythmic drug therapy for suppression of ventricular premature beats is no longer recommended. Suppression with anti arrhythmic drug therapy may lead to risk of sudden death due to severe bradycardia and asystole.

When ventricular premature beats are associated with sinus tachycardia may be due to sympathetic stimulation and adrenergic drive and can be treated by a β blocker.

Accelerated idio ventricular rhythm:

About 20% of the patients exhibit this type of rhythm. It occurs within 2 days of acute myocardial infarction and often occurs transiently during fibrinolytic therapy.

It is benign and it does not lead to the development of classic venricular tachycardia and hence no treatment is needed.

Ventricular tachycardia:

Ventricular tachycardia – non sustained or sustained and polymorphic ventricular tachycardia can occur during the acute phases of ischaemia and infarction.

Ventricular tachycardia can also occur during reperfusion. Non sustained or sustained VT which often results from re-entry can occur late after MI.

Short term treatment of ventricular tachycardia after acute MI depends on the hemodynamic status of the patient and the presence of an on going or recurrent ischemia.

Non sustained Ventricular Tachycardia

- Usually do no require any treatment

Sustained Ventricular Tachycardia

Haemodynamically stable

- IV amiodarane (bolus of 150 mg over 10 min, followed by infusion of 1.0 mg / mt for 6 hours and then 0.5mg / min)

- Procainamide (bolus of 15mg/min over 20 – 30 min; infusion of 1 – 4 mg / min)

Haemodynamically unstable

- Cardioversion - unsynchronized discharge of 200 – 300 J (monophasic)
- Patients who are refractory to electroshock should be treated with epinephrine (1 mg IV) before cardioversion.

Torsedes despointes:

Ventricular arrhythmias including the unusual form of ventricular tachycardia known as torsedes des pointes may occur in patients with STEMI. Polymorphic VT patients who demonstrate a long QT interval during their baseline rhythm typically is referred to as torsades despointes. Secondary causes like electrolyte disturbances, hypoxia, drug intake (quinidine or digoxin) should be identified and treated first.

Ventricular fibrillation:

After acute myocardial infarction VF can occur in 3 settings.

- Primary ventricular Fibrillation – occurs suddenly without any premonitoring signs in patients with MI.
- Secondary ventricular fibrillation – associated with left ventricular failure and cardiogenic shock.

- ❖ Late ventricular fibrillation – occurs after 48 hours of acute myocardial infarction and it frequently occurs in patients with large infarcts and ventricular dysfunction.[42]

Treatment:

- ❖ Prophylaxis with lidocaine after acute myocardial infarction to prevent primary ventricular fibrillation is now a days not recommended.
- ❖ Unsynchronized electrical countershock with at least 200 – 300 joules should be given for patients with ventricular fibrillation after acute myocardial infarction.

Brady arrhythmias:

Sinus bradycardia

It is the common type of arrhythmia in the early hours of acute myocardial infarction especially in patients with inferior wall and posterior wall myocardial infarction. The mechanism of sinus bradycardia is due to increased vagal tone, it may be actually protective because it reduces myocardial oxygen demand. So the mortality rate is lower in patients with this type of arrhythmia.[46]

Treatment:

- If the patient is hemodynamically stable and without ventricular ectopy no treatment is needed.
- Haemadynamically unstable – Inj Atropine 0.3 – 0.6mg IV every 3 – 10 min (total dose not exceeding 2mg) to bring the heart rate upto approximately 60 beats / min.

Atrio ventricular and intra ventricular conduction disturbances:

The mechanism of conduction block at any level of atrioventricular or intra ventricular conduction system is due to ischaemic injury to the conduction system.[47]

The mortality rate of patients who develop AV block in anterior wall myocardial infarction are higher than that of patients in inferior wall myocardial infarction. Because in inferior wall infarction the heart block is commonly due to increased vagal tone and due to release of adenosine and therefore, it is transient.

Treatment:

First degree AV block. Generally does not require specific treatment. If it is associated with sinus bradycardia and hypotension Inj.atrophine. 0.3 – 0.6 mg iv every 3-5 min can be given (maximum dose upto 2mg).

Second degree AV block:

If haemodynamically unstable Inj. atropine (0.3 – 0.6mg) is indicated.

Type II second degree AV block usually arises from injury of the conduction system below the His bundle. It has high predilection to go for complete heart block so temporary external or transvenous pacemake should be inserted with rate set at 60 beats / min.

Complete heart block:

In inferior wall infarction complete heart block is usually transient in nature so treatment is usually not indicated.

But if the patient is haemadynamically unstable (ventricular arrhythmias, hypotension, pumpfailure) in anterior wall myocardial infarction, temporary transvenous pacing protects against asystole in patients with complete heart block.[48]

RIGHT BUNDLE BRANCH BLOCK:

RBBB can alone lead to AV block because often it is a new lesion usually associated with Anteroseptal infarction. Isolated right bundle branch block is associated with increased mortality rate in patients with anterior wall myocardial infarction if it is associated with congestive heart failure.

BIFASCICULAR BLOCK;

The combinations of right bundle branch block with either left anterior or posterior divisional block or the combination of left anterior and posterior divisions of the left bundle branch is known as bidivisional or bifascicular block.

Bifascicular block in the presence of prolongation of PR interval (first degree AV block) may indicate disease of the third subdivision rather than that of the AVnode and is associated with a greater risk of complete heart block.[50]

Assessment for electrical instability in the post infarction period:

There is a greater risk of sudden cardiac death due to malignant ventricular arrhythmias can occur.

Measures to stratify the patients who are at increased risk of sudden death includes[51]

1. Measurement of Q-T dispersion (variability of Q-T intervals between ECG leads).
2. Ambulatory ECG recordings (Holter monitoring)
3. Invasive electro physiological testing.
4. Recording a signal averaged electro cardiogram. (SAECG).SAECG reveals the presence of late potentials that are low in amplitude

high frequency waveforms within the terminal portion of the QRS complexes.Late potentials reflect the presence of slow conduction within the ventricular myocardium which acts as an arrhythmogenic site.It is mainly due to fibrosis of the injured myocardium in between the areas of viable myocardium.

5. Measuring heart rate variability (beat – beat variability in R-R interval)
6. Baroreflex sensitivity (beat to beat change in sinus rate in response to blood pressure changes).

Prophylactic anti arrhythmic therapy:[51]

There are three large post infarction trial studies have conducted to prove that the control of ventricular and atrial arrhythmias can be done by prophylactic anti arrhythmic drug therapy after STEMI or not.

Cardiac arrhythmia suppression Trial (CAST) tested with encainide, flecainide or morizicine were studied. The study was stopped prematurely because increased mortality rate was observed in patients in the treatment group.

Another trial was done with oral D-sotalol (survival with oral D-sotalol or SWORD). In patients with depressed LV function (EF < 40%) D-sotalol was given as a prophylaxis for arrhythmias. This study was also

stopped prematurely because of observation of increased mortality in treatment group.

Another study the Canadian Amiodarone Myocardial Infarction Trial (CAMIAT) showed that amiodarone reduced the incidence of VPDS in patients with recent H/o acute myocardial infarction.

European Amiodarone Myocardial Infarction Trial (EMIAT) also showed reduction in the mortality of patients after acute MI.

NO-REFLOW:[52]

In patients with Acute myocardial infarction myocardial reperfusion does not occur even after successful reopening of infarct related coronary artery which is called No-Reflow phenomenon. Depending on the type of reperfusion therapy about 10-40% of the patients show evidence of No-Reflow.It is important because it causes prolonged myocardial ischaemia which is associated with increased risk of severe arrhythmias and hemodynamic instabity.

Treatment for No-Reflow include intracoronary administration of vasodilators like adenosine, verapamil, nicorandil, papaverine and nitroprusside.

Localization of the culprit vessel in ECG:

ECG is the most (cost) effective and feasible diagnostic tool in cardiac evaluation. ECG localization of the deceased coronary artery in

STEMI during admission aids in early initiation of aggressive therapy and prognostication.

ECG diagnosis of STEMI is made by:

1. New ST elevation at J point in two contiguous leads
2. CUT – OFF points for ST elevation in V2-V3

   > 2mm - men

   > 1.5mm – women

   Other leads - > 1MM

STEMI can be subendocardial or epicardial

Regional ST segment changes in STEMI

1. Anterior Wall – ST elevation in V1 – V4
2. Lateral wall – ST elevation in I, aVL, V5, V6
3. Inferior wall – ST elevation in II, III, aVF
4. RV MI – ST elevation in V4 R
5. Posterior wall – ST elevation in V8, V9 with ST depression in V1 – V3.

Regional ST elevation and occluded Coronary Artery

Anterior wall MI - Left anterior descending coronary artery (LAD) or left main coronary artery (LMCA)

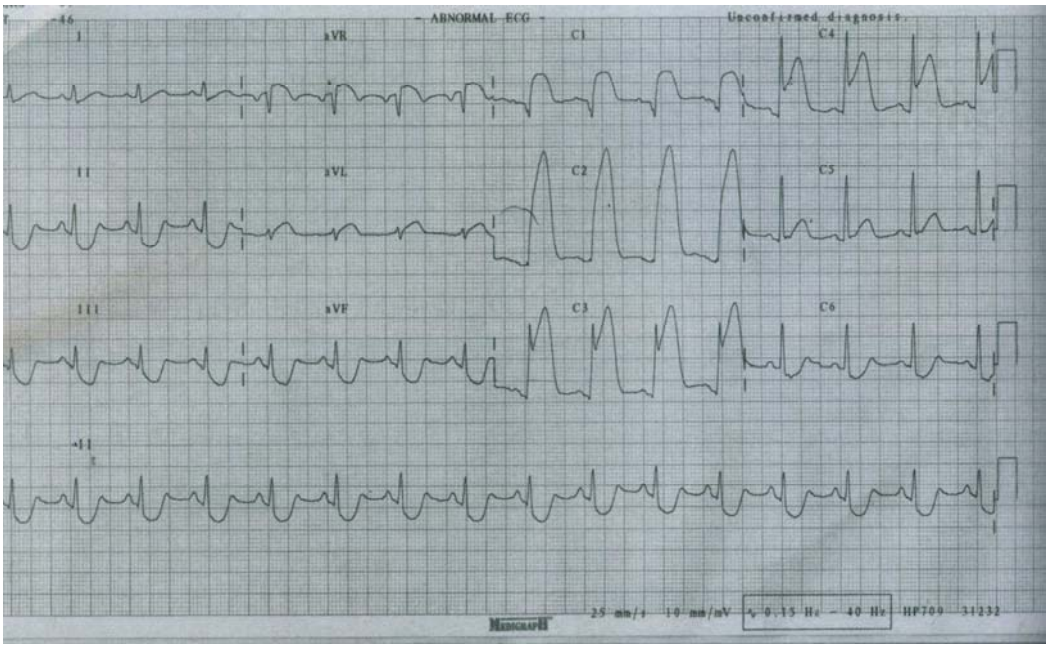
Inferior wall MI – Right Coronary Artery (RCA) or left circumflex artery (LCX)

Lateral wall MI – Left circumflex artery (LCX)

Localisation of anterior wall STEMI

Anterior wall STEMI is diagnosed by ST elevation in V1 – V4 and occurs due to occlusion of LAD artery at different levels

1. In proximal LAD disease this occlusion is proximal to S1 and D1 (First septal and diagonal branches). At this level, the basal IVS and basal LV is injured and the vector is directed superiorly and to the right.

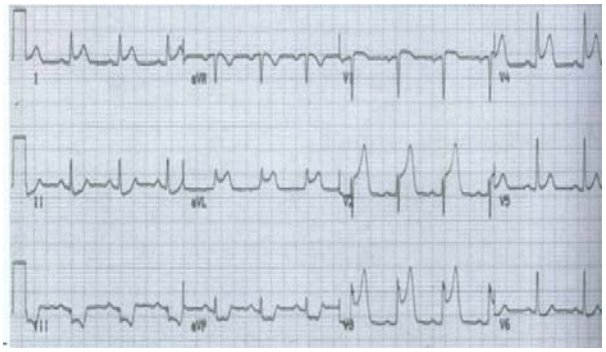


Proximal LAD disease

PROXIMAL LAD LESION: (Proximal to S1 and D1) – ECG changes

- ST elevation in aVR and V1 with elevation V1>aVR
- ST elevation in V2V3V4
- ST depression (Reciprocal) in LII, L III, avF, V5, V6
- Acute onset RBBB may be seen
- These patients belong to High risk category

2. In mid LAD disease, the occlusion is distal to S1 and proximal to D1 with the basal IVS being injured and basal LV being spared and the vector is directed superiorly and to the right.

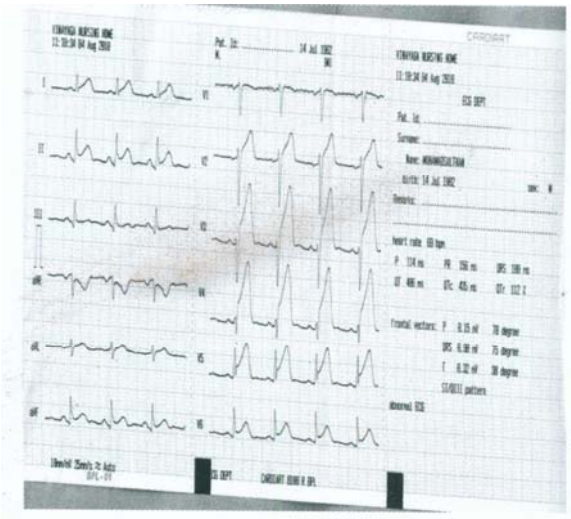


MID LAD OCCLUSION

MID LAD OCCLUSION: (distal to S1 and proximal to D1) – ECG changes

- No ST elevation in avR or V1
- ST elevation in V2 to V6
- Reciprocal ST depression LII, L III, avF
- No RBBB
- These patients belong to intermediate risk

3. In distal LAD disease, the occlusion is distal to S1 and D1 with both the basal IVS and LV being spared and the vector is directed inferiorly and to the left.



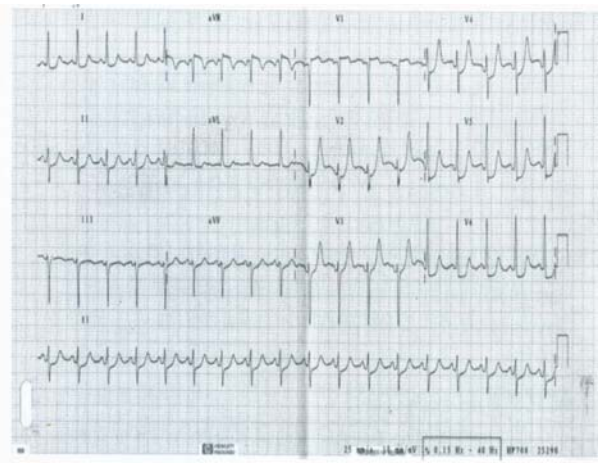
DISTAL LAD DISEASE

DISTAL LAD OCCLUSION (distal to S1 and D1) – ECG changes

- ❖ No ST elevation in avR or V1
- ❖ ST elevation in V2 to V6
- ❖ No reciprocal ST depression
- ❖ No RBBB

4. Left Main Coronary Artery (LMCA) occlusion – ECG changes

- ❖ ST elevation in avR > V1 with predominant ST depression in anterior chest leads



Left Main Coronary Artery

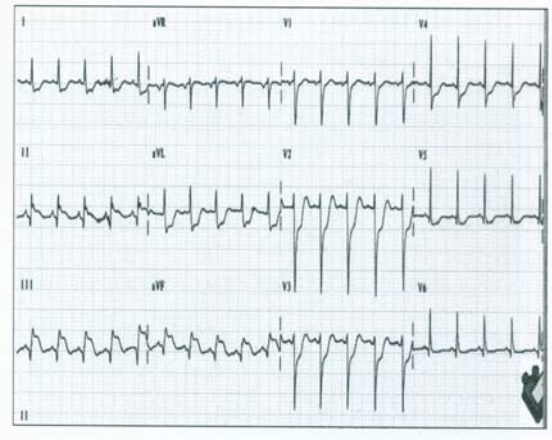
Localization of IWMI/RVMI/PWMI:

Inferior wall MI is diagnosed by ST elevation in II, III, aVF and associated RV infarction by elevation in right sided V4. Posterior wall MI is diagnosed by ST elevation in V8, V9 and ST depression in V1-V3. occlusion may be in the RCA artery or LCX artery for IWMI and it is in the LCX artery for PWMI.

Right coronary artery occlusion – ECG changes

a. Proximal RCA:

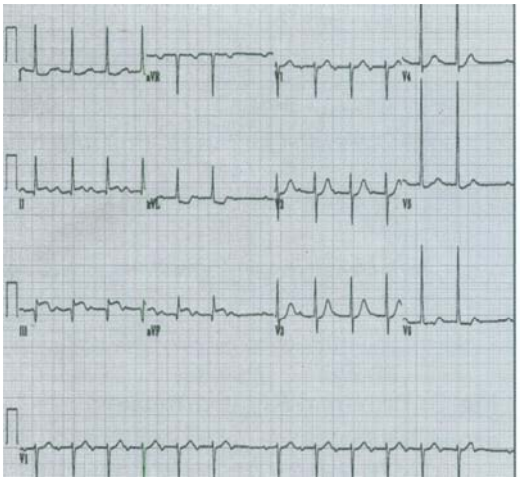
- Inferior Wall MI with RVMI and PWMI
- ST elevation in II, III, aVF with elevation L III > L II
- Associated ST depression in V1 – V2 and discordant (ie) ST depression V2 > V1
- V4 R ST elevation indicates associated RV infarction



Proximal RCA Occlusion

b. Distal RCA (only IWMI) – ECG changes

- ST elevation L III > L II
- No ST depression in V1 – V3 (no PWMI)
- No ST elevation in V2 (no RVMI)



Distal RCA Occlusion

c. Left Circumflex Artery – ECG changes

- ST elevation L II > L III
- ST depression in LI and aVL with discordance aVL > LI
- ST depression in V1 - V3 if posterior wall MI is present

ECG localization of Coronary artery occlusion in Acute STEMI helps in early aggressive medical management and in planning early revascularization. Two important factors should be kept in mind while analyzing the ECG in STEMI

i. Look at inferior leads in anterior wall MI and anterior leads in inferior MI

ii. Look at aVR and V1 (the often neglected leads)

Hence, in a way ECG can be considered as poor man's coronary angiogram if interpreted skillfully.

NONARRHYTHMIC COMPLICATIONS OF MI;

1. Ventricular septal rupture & Ventricular free wall rupture
2. Papillary muscle rupture and mitral regurgitation.
3. Recurrent infarction & ischaemia.
4. Pericardial effusion and pericarditis.(Dresslers syndrome)
5. Venous thrombosis and pulmonary embolism.
6. Left ventricular aneurysm.
7. Left ventricular thrombus and Arterial embolism

CARDIOGENIC SHOCK:

In patients with Acute myocardial infarction cardiogenic shock is the leading cause of death. Characterized by systolic arterial BP < 90 mmHg , cardiac index < 2.2 L/ min/m^2 and an elevated filling pressures pulmonary capillary wedge pressure (PCWP) > 18 mmHg. The inhospital mortality rate is high that is > 50%.

Cardiogenic shock is more common in anterior wall myocardial infarction especially associated with risk factors like old age group , female sex, prior myocardial infarction , diabetes mellitus are more prone for cardiogenic shock. Shock associated with first attack of inferior wall myocardial infarction should prompt a search for a mechanical cause.

Women with AMI:

Women with myocardial infarction have a 56% risk of early mortality after a first attack of myocardial infarction than that of in men.

- Presentation is with atypical manifestations.
- More common in women above 65 years of age.

So High index of suspicion is needed for diagnosis. Common presentations are

- Breathlessness
- Abdominal pain
- Sweating and syncopal attacks.

- ❖ Both arrhythmic and non-arrhythmic complications are high.
- ❖ Mortality rate is higher in atypical MI than that in typical MI.

MI in elderly:

- ❖ Most of the elderly people presents with atypical manifestation.
- ❖ Mortality rate in elderly patients with Myocardial infarction was higher than that in younger patients with Myocardial infarction.
- ❖ Risk of arrhythmias are more common in the elderly population when compared to younger population.

MATERIALS AND METHODS

100 consecutive patients with AMI admitted within 1 hour of chest pain to the intensive care unit of Tirunelveli Medical College Hospital were studied over a period of 12 months from October 2011 to October 2012.

All patients admitted to ICCU with clinical features and ECG evidence suggestive of acute Myocardial Infarction are taken. They were subjected to continuous ECG monitoring and Echo cstudy. Results were taken up for study.

INCLUSION CRITERIA:

- All patients with clinical features and ECG evidence suggestive of acute Myocardial Infarction.
- Presence of arrhythmias pre & post thrombolysis.

EXCLUSION CRITERIA:

- Patients with out Myocardial Infarction showing arrhythmias.
- Patients with previous history of myocardial infarction, previous cardiac surgeries and those who have been treated outside and valvular heart diseases patients were excluded from the study.

The diagnosis of AMI was made with

1. History:

Classical chest pain retrosternal heavy, squeezing and crushing although occasionally it is described as stabbing or burning, commonly occurs at rest. Usually more severe and last longer and it radiates to the arms.

Electro cardiogram:

Myocardial injury is reflected electrocardiographically by deviation of the ST segment. The ST segment is deviated towards the surface of the injured tissue.

The characteristic feature of the hyperacute phase of myocardial infarction is slope elevation of the ST segment and it is taken as the definite evidence of myocardial infarction and type of arrhythmias were also noted.

Biochemistry:

Cardiac biomarkers

- Ratio (relative risk) of CK-MB mass to CK activity of about 2.5 was taken as an indicator of myocardial injury.
- Troponin T (TnT) by cord test assay was done in all patients.

Other investigations like complete blood count, serum electrolytes, blood sugar, urea, creatinine and SGOT / SGPT level was done in all patients.

Physical examination:

Immediately after admission a complete physical examination was done which includes.

- Pulse rate, rhythm, character, volume,condition of the vessel wall and examined in all peripheral sites.
- Blood pressure in both upper limbs.
- Jugular venous pressure
- Apical impulse position and character
- Auscultation of heart sounds, murmurs, additional sounds.
- Respiratory rate and added sounds in both lung fields were noted.
- Fundus examination was done in all patients.

Risk factor analysis:

Obesity:

Body mass index of >30kg/m^2 was considered as obese.

Smoking:

Patients who are smoking for more than 6 months and more than 10 cigarettes / day were considered as smokers.

Systemic HT:

BP was recorded in all patients and BP > 140/90 mm Hg on more than 2 occasions or with a history of systemic HT with end organ damage like retinopathy, nephropathy etc were considered as hypertensives.

Diabetes Mellitus:

Blood sugar level more than 126 mg/dl in the fasting state and more than 200mg/dl in the post prandial state were considered as a diabetic.

Family history and personal history:

Family history of coronary artery heart disease and family history of sudden death were considered as positive.

H/o alcoholism was elicited.

In all female patients presented with acute myocardial infarction menstrual history and menopaused status was recorded.

ECHOCARDIOGRAM:

Echocardiogram was done in all patients to look for regional wall motion abnormalities and to rule out aotic dissection in patients presented with chest pain and non diagnostic ECG.

Sinus tachycardia:

Maximum recorded sinus rate is 220 beats/min – age.

Normal 'P' wave is followed by normal 'QRS' complexes.

PR interval is shorter than during normal sinus rhythm.

Supra ventricular tachycardia:

Atril fibrillation:

- ❖ Irregularly irregular QRS complexes.
- ❖ Chaotic baseline – No clear 'P' waves
- ❖ QRS complex looks normal, or the same as when the patient is in sinus rhythm.

Atrial flutter:

- ❖ Saw tooth appearance of baseline.
- ❖ Ventricular rate can be fast, normal or slow and may be regular or irregular.
- ❖ Presents as in a ratio of 2:1, 4:1, 6:1, 8:1.
- ❖ Flutter waves are best seen in standard lead II and lead V1.

Junctional ectopic beats:

- ❖ Normal sinus beats are interspersed with QRS complexes occurring earlier than expected.
- ❖ Premature QRS complexes having the same morphology as in sinus rhythm but are not proceeded by a 'P' wave.

- There may be a 'P' wave immediately after the QRS complex or buried in the QRS complex itself.
- The following normal sinus beat occurs later than expected.

Ventricular arrhythmias:

Ventricular premature beats

- Premature, wide and bizarre QRS complexes
- Opposite to the direction of QRS - ST, T wave changes appear
- Before the premature QRS complexes no premature `P' wave
- The pause is fully compensatory after the VPD
- `R' wave of the VPD on the apex of `T' wave (R on T phenomenon)
- The VPD may fuse with subsequent QRS complex
- The compensatory pause may be short if the VPD captures the SA node.

Interpolated ventricular extra systoles

- Extra systole which is sandwiched between two conducted sinus beats occurs without a compensatory pause.
- The sinus beat following the extra systole has a longer PR interval than the sinus beat proceeding the extrasystole.

Ventricular bigeminy

Extra systoles which occur after every other sinus beat are the commonest cause of bigeminal rhythm.

Multifocal or multiform ventricular extrasystoles.

Extra systoles that arise from different foci and consequently give rise to different QRS complexes.

Extra systoles in pairs

When a ventricular ectopic focus discharges prematurely and twice in succession the rhythm will manifest as a pair of extrasystoles – called couplets.

Extra systolic paraoxysmal ventricular tachycardia

Three or more successive ventricular extrasystoles constitute an extra systolic ventricular tachycardia

VPC's are graded by lown et al, because they may predispose to ventricular fibrillation.

Grade	O	- No VPC's
Grade	1	- VPC's < 30 / hour
Grade	II	- VPC's > 30 / hour
Grade	III	- VPC's which are multifocal
Grade	IV-A	- Bigeminy / coupled VPC's
Grade	IV-B	- Salvos of 3 or more

Grade V - R on T phenonena

1. Ventricular tachycardia:

Extra systolic ventricular tachycardia is a series of three or more consecutive ventricular extra systoles.

Ventricular capture:

A sinus impulse may reach the AV node during a non-refractory phase. The sinus impulse can than be conducted to the ventricles and momentarily activate or capture the ventricle (ie) for one beat only. This conducted beat which occurs during the ectopic ventricular rhythm is known as a capture beat.

Ventricular fusion:

At times the capturing (sinus) impulse may invade the ventricles concomitantly with the ectopic ventricular impulse. The QRS complex will have a configuration that is in between that of the pure sinus beat and pure ectopic beat.

Accelerated idioventricular rhythm

- ❖ bizarre QRS complexer or ventricular fusion beats
- ❖ Rate is in the range of 70 – 80 beats / min.
- ❖ Propensity to AV dissociation and capture beats.
- ❖ The absence of pacemaker protection.

Ventricular flutter:

- ❖ A very rapid and regular ectopic ventricular discharge > 200 / mt.
- ❖ Grossly abnormal intraventricular conduction.
- ❖ Mutiform ventricular flutter, torsades depointes

(Multiform QRS complexes, a manifestation which has been termed torsedes de pointes – a twisting or torsion at points)

Ventricular fibrillation:

Completely irregular, chaotic and deformed deflexions of varying height, width and shape. Regular waveforms such as p waves. QRS complexes, ST segments and T waves cannot be identified.

Conduction disturbances:

SA block:

- ❖ This resembles the slow regular rhythm of sinus bradycardia.
- ❖ Subsequent beat may be a normal sinus beat, an AV nodal escape beat or a ventricular escape beat.
- ❖ In second degree SA block neither the p wave nor the QRS complex is recorded at the moment of block.

AV block:

- ❖ I^0 AV block – is a delay in conduction through the conducting system. It if reflected by a prolonged P-R interval.
- ❖ II^0 AV block – intermittently the `P' wave is not followed by a QRS complex and a ventricular beat is so to speak ` dropped'.
- ❖ III^0 AV block Characterized by
    i. AV dissociation
    ii. Slow ventricular rate
    iii. QRS configuration is normal or near normal in shape.

Criteria for Right Bundle Branch Block:

1. Lead V1 reflects a tall, wide, and frequently notched R' deflextion.
2. The left oriented leads, leads V5 and V6 as well as standard lead I reflect a prominent, delayed and widened S wave.
3. The QRS duration is increased to 0.14 sec or longer.

Criteria for Left Bundle Branch Block:

1. The QRS complex is prolonged to 0.12 sec or more and may be as long as 0.20sec.
2. aVL
    - ❖ An RSR complex
    - ❖ A wide but unnotched complex.

3. V1 and V2 – Widened, notched QS complex or an rS complex.
4. The ST segment and `T' wave are opposite in direction to the terminal QRS deflexion.

Left anterior fasicular block:

- ❖ Left axis deviation of the mean manifest frontal plane QRS axis.
- ❖ Increased ventricular activation tine.
- ❖ Secondary `T' wave repolarisation changes.
- ❖ Slight slurring or irregularity of the QRS limbs.
- ❖ Increased magnitude of the dominant QRS deflexion.

Left posterior hemiblock:

Manifested by

1. Right axis deviation of the mean manifest frontal plane QRS axis.
    - Prominent `S' waves in strandard lead I and lead AVL.
    - Tall R waves in standard II and III and lead AVF.
2. Mean QRS forces are increased in magnitude.
3. The distal limb of the tall `R' wave in standard lead III is frequently notched or slurred.

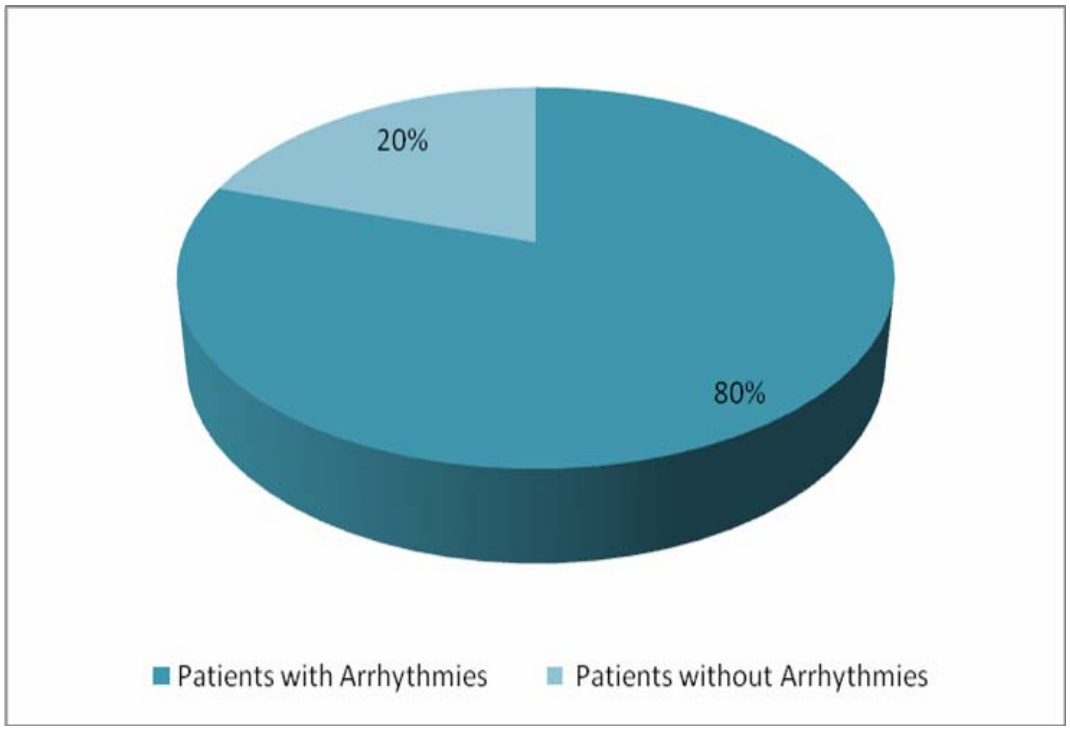
RESULTS AND OBSERVATIONS

Total number of patients with acute myocardial infarction under study =100

Table 1

ARRHYTHMIES IN AMI

No. of Patients with Arrhythmias	No. of Patients without Arrhythmias
80%	20%



80% of the patients with acute myocardial infarction had arrhythmias.

Table 2

SEX DISTRIBUTION OF AMI

MALE	FEMALE
73	27

Total no of males with acute myocardial infarction - 73

Total no of females with acute myocardial infarction - 27

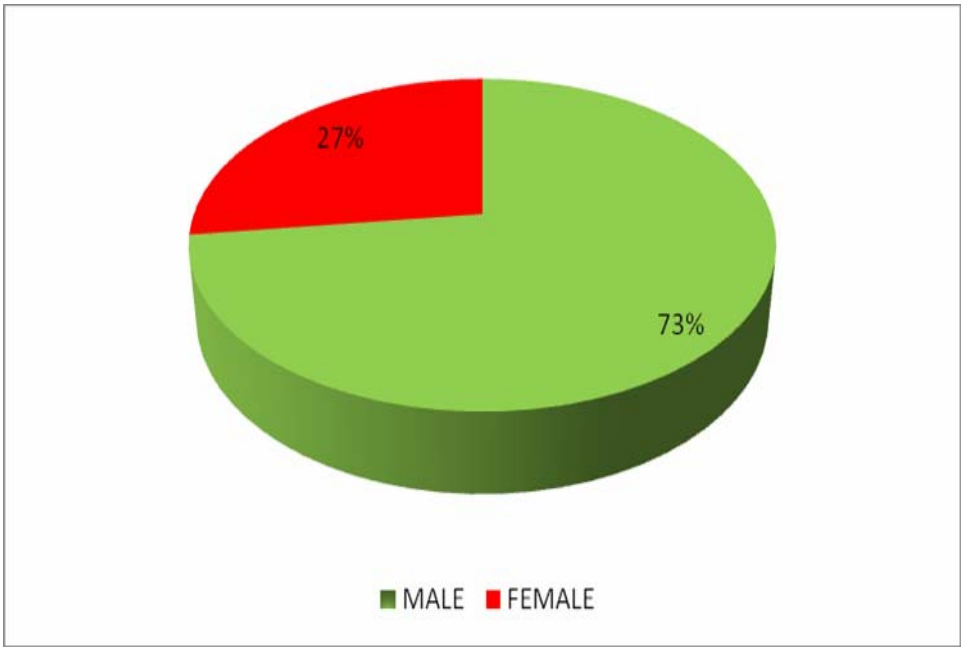


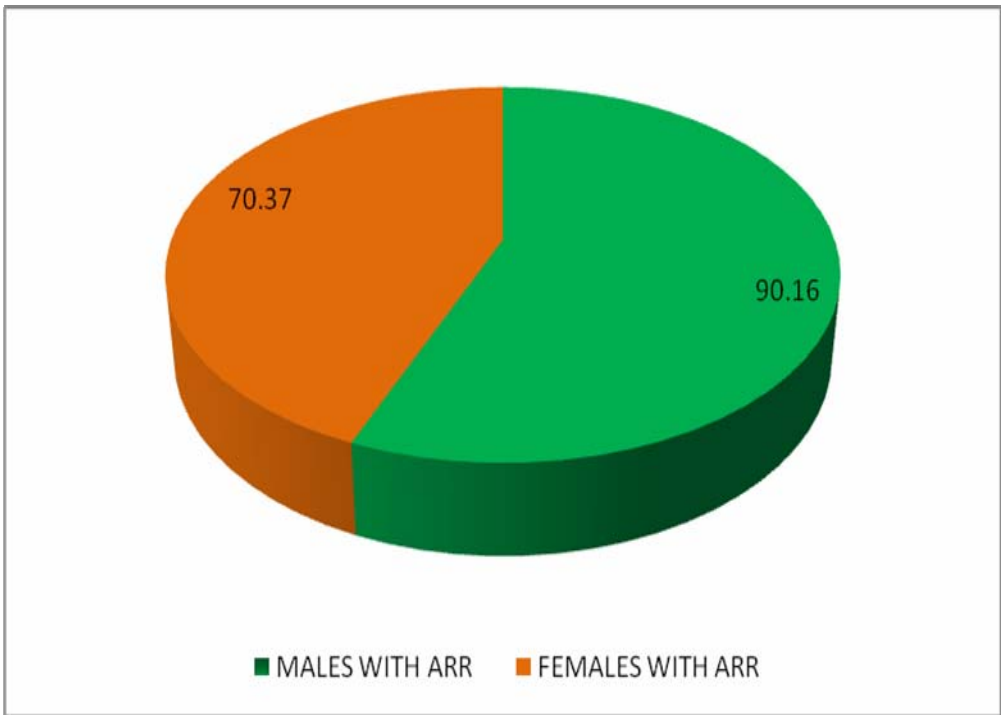
Table 3

SEX DISTRIBUTION OF ARRHYTHMIAS IN AMI

No. of MALES with ARR	% of males	No. of FEMALE with ARR	% of FEMALES
61	90.16%	19	70.37%

Total no of males with acute MI with arrhythmias – 61 (90.16%)

Total no of females with acute MI with arrhythmias – 19 (70.37%)

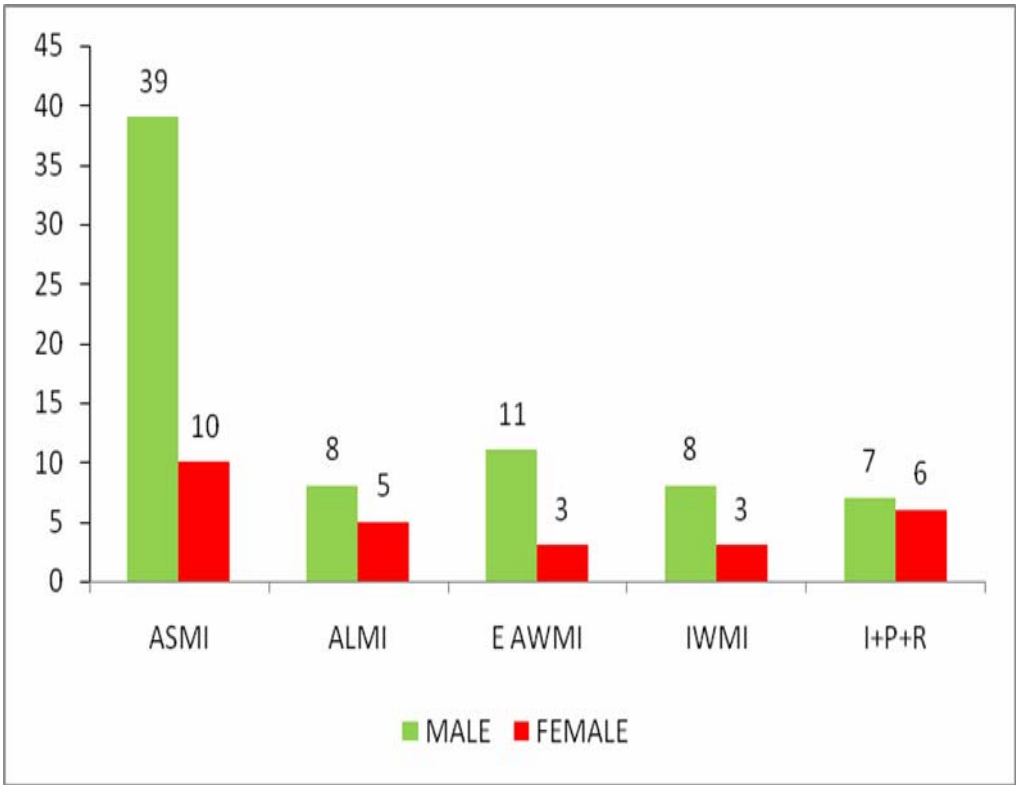


Study shows that males had higher incidence of acute MI and the resultant arrhythmias

Table 4

SEX DISTRIBUTION IN RELATION TO LOCATION OF MI

SEX	ASMI	ALMI	E AWMI	IWMI	I+P+R
MALE	39	8	11	8	7
FEMALE	10	5	3	3	6
TOTAL	49	13	14	11	13



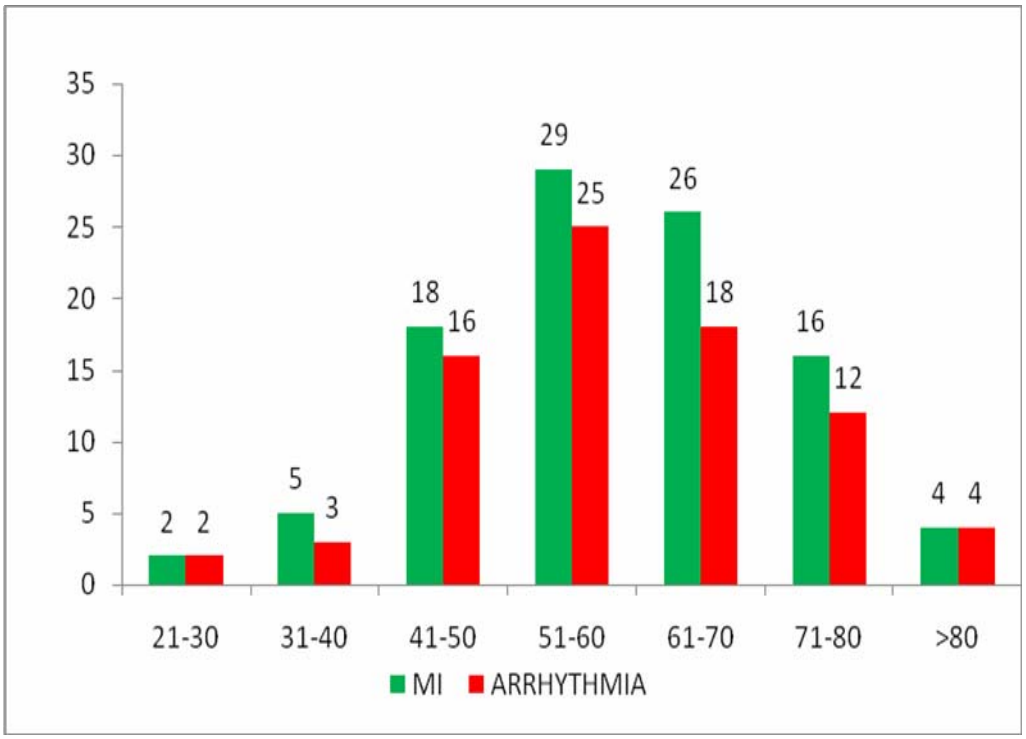
Anterior wall MI (especially ASMI) was the most common presentation.

Males had higher incidence of acute MI

Table 5

AGE & SEX DISTRIBUTION AND INCIDENCE OF ARRHYTHMIAS

AGE GROUP	INCIDENCE OF MI (NO.OF PATIENTS)			INCIDENCE OF ARRHYTHMIAS %		
	MALE	FEMALE	TOTAL	MALE	FEMALE	TOTAL
21-30	2	-	2	2(100%)	-	100%
31-40	5	-	5	3(60%)	-	60%
41-50	16	2	18	14(87.5%)	2(100%)2	88.88%
51-60	26	3	29	23(88.49%)	2(66.66%)	79.31%
61-70	13	13	26	9(69.23%)	9(69.23%)	69.23%
71-80	9	7	16	7(77.77%)	5(71.42%)	75.00%
>80	2	2	4	2(100%)	2(100%)	100%
TOTAL	73	27				



Oldest patient recorded – 85 years

Youngest patient recorded – 29 years

Highest incidence of MI was observed in the age group 51 – 60 years in males and 61 – 70 years in females.

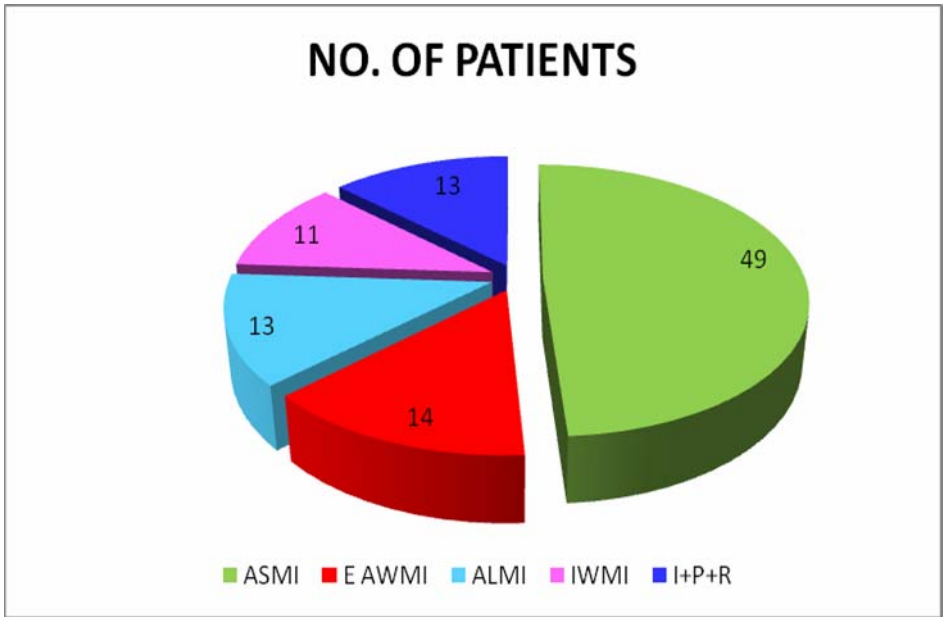
Highest incidence of arrhythmias are noted in the age group of 41 – 50 years mainly due to sinus tachycardia.

2 patients <30 years arrhythmias which were benign on the other spectrum 2 patients above 80 years had arrhythmias which were malignant (one patient had complete heart block, other had ventricular fibrillation).

Table 6

SITE OF INFARCTION AND INCIDENCE OF ARRHYTHMIAS

S.NO	SITE OF INFARCTION	NO. OF PATIENTS	% OF ARRHYTHMIAS
1	ASMI	49	75.8%
2	E AWMI	14	85.7%
3	ALMI	13	53.84%
4	IWMI	11	81.88%
5	I+P+R	13	84.61%

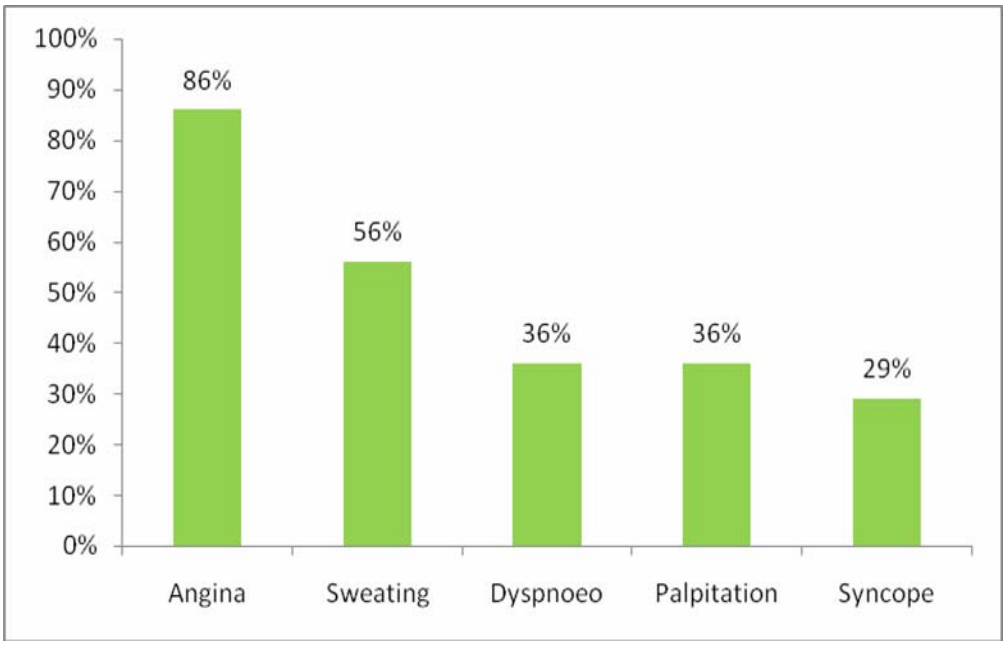


Extensive anterior wall of MI was associated with higher incidence of arrhythmias 85.7%. Inferior wall MI also showed about 81.88% arrhythmias when sinus bradycardia is included.

Table 7

SYMPTOM ANALYSIS

SYMPTOM	%
Angina	86%
Sweating	56%
Dyspnoea	36%
Palpitation	36%
Syncope	29%

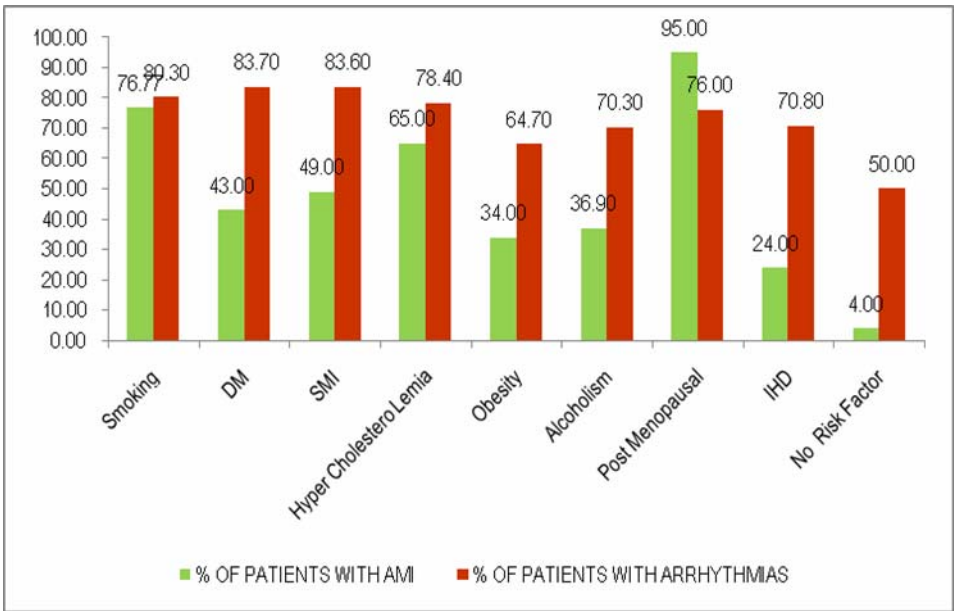


Among 100 patients 86% of patients were admitted with classical angina and 14 with angina equivalents.

Table 8

RISK FACTOR ANALYSIS SINGLE RISK FACTOR

Risk Factors	% of Patients with AMI	% of Patients with Arrhythmias
Smoking	76.77%	80.3%
DM	43%	83.7%
SHT	49%	83.6%
Hyper Cholesterolemia	65%	78.4%
Obesity	34%	64.7%
Alcoholism	36.9%	70.3%
Post Menopausal	95%	76%
IHD	24%	70.8%
No major Risk Factor	4%	50%

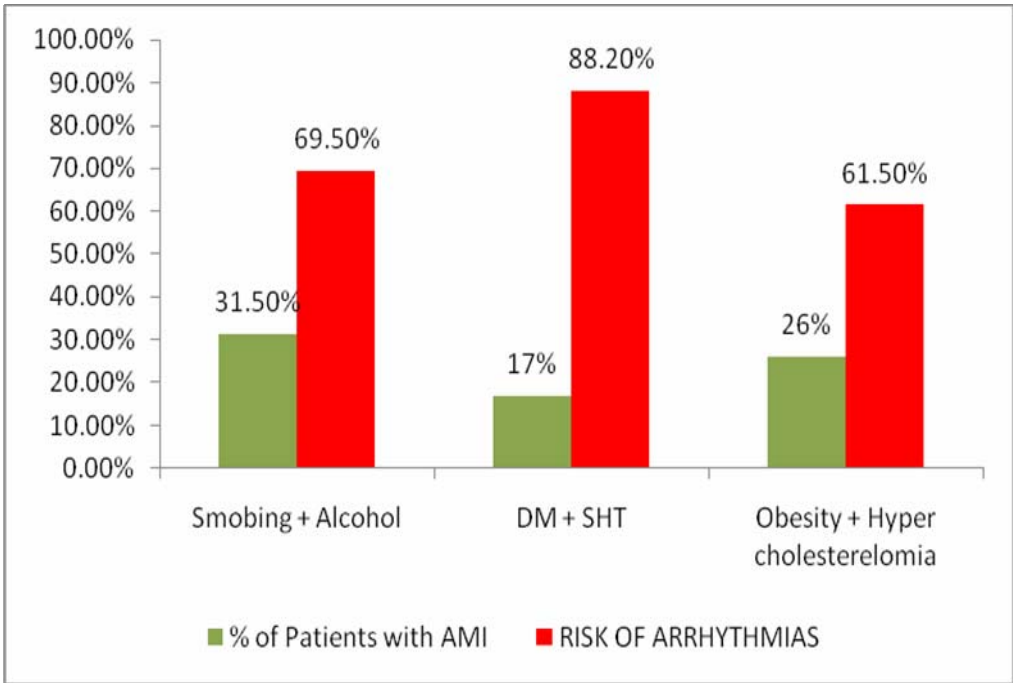


76% of patients with acute MI had smoking as a major risk factor but incidence of arrhythmias are almost equal in risk factors like smoking(80.3%), DM (83.7%), SHT (83.6%) and hypercholesterolemia (78.4%)

Table 9

MUTIPLE RISK FACTORS

MULTIPLE RISK FACTORS	% of Patients with AMI	RISK OF ARRHYTHMIAS
Smoking + Alcohol	31.5%	69.5%
DM + SHT	17%	88.2%
Obesity + Hyper cholesterolemia	26%	61.5%

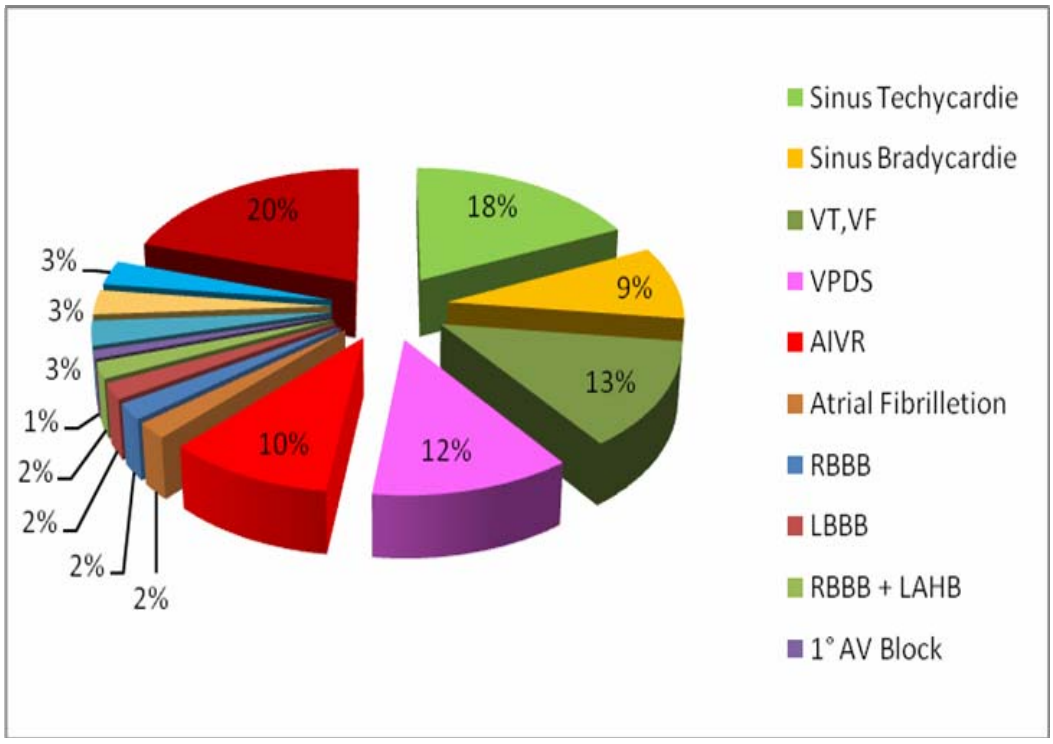


Incidence of acute MI is more common in patients with risk factors like Smoking and alcohol (31.5%). But incidence of arrhythmias are more common in patients with risk factors like DM + SHT (88.2%)

Table 10

Distribution of Arrhythmias in Patients with AMI

S. No	ARRHYTHMIA	% OF CASES
1	Sinus Tachycardia	18%
2	Sinus Bradycardia	9%
3	VT,VF	13%
4	VPDS	12%
5	AIVR	10%
6	Atrial Fibrillation	2%
7	RBBB	2%
8	LBBB	2%
9	RBBB + LAHB	2%
10	1° AV Block	1%
11	2° AV Block	3%
12	BFB	3%
13	CHB	3%
14	NO Arrhythmias	20%

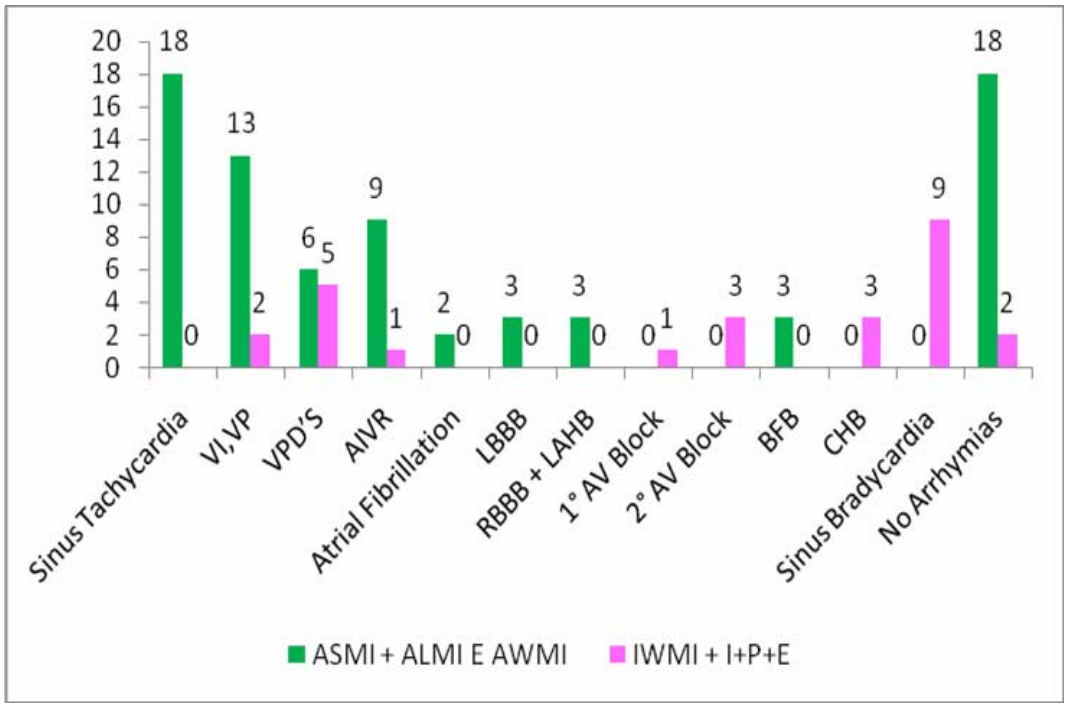


Sinus tachycardia is the most common type of arrhythmias (18%) next common type is VT + VF (13%). Next most common type are VPDS

Table 11

ARRHYTHMIAS IN RELATION TO LOCATION OF MI

S. No	Arrhythmias	ASMI + ALMI E AWMI	IWMI + I+P+E
1	Sinus Tachycardia	18(31.03%)	-
2	VT,VF	13(22.41%)	2(9.09%)
3	VPD'S	6(10.34%)	5(22.72%)
4	AIVR	9(15.51%)	1(4.04%)
5	Atrial Fibrillation	2(0.34%)	-
6	LBBB	3(5.17%)	-
7	RBBB + LAHB	3(5.17%)	-
8	1° AV Block	-	1(4.54 %)
9	2° AV Block	-	3(13.63%)
10	BFB	3(5.17%)	-
11	CHB	-	3(13.63%)
12	Sinus Bradycardia	-	9(40.90%)
13	No Arrhythmias	18(23.68%)	2(8.33%)



AWMI + ALW = 58(76.31%)

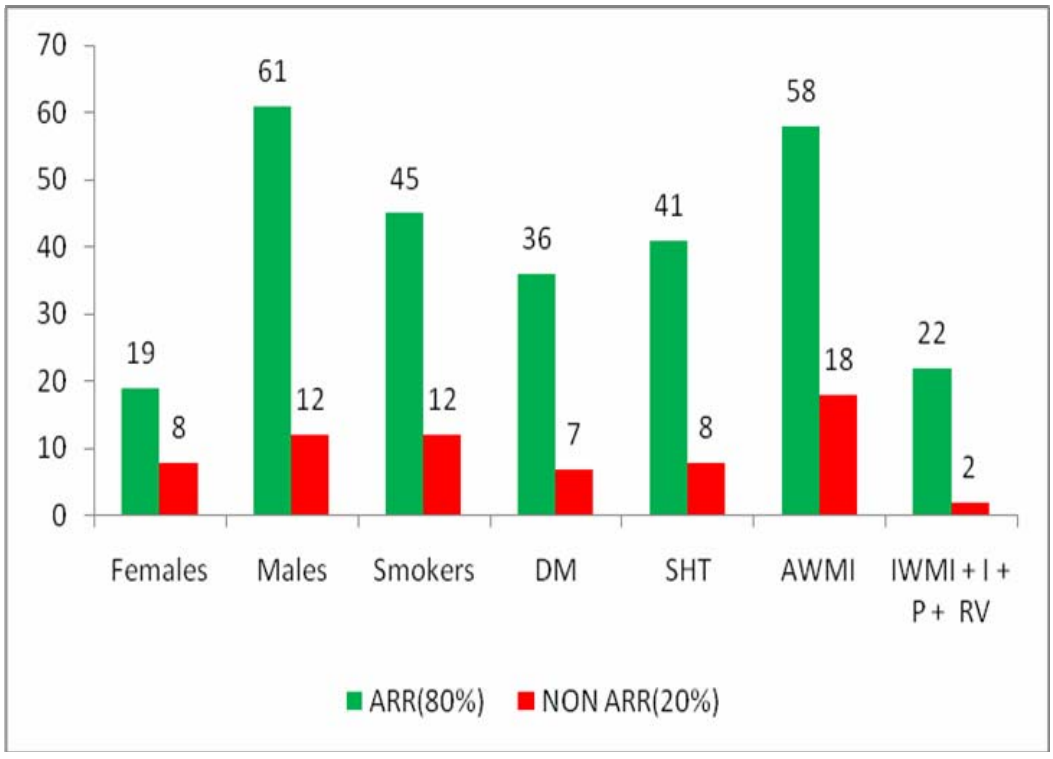
IWMI+I+P+R = 22(91.66%)

Overall incidence of arrhythmias were higher in IWMI (91.66) when compared with AWMI (76.31%) when sinus bradycardia was included in IWMI patients. In AWMI sinus tachycardia was most common. In IWMI sinus bradycardia was most common.

Table 12

SIGNIFICANCE OF ARRHYTHMIAS

S. No	Character	ARR(80%)	NON ARR(20%)	P Value
1	Females	19(70%)	8(29.6%)	<0.01
2	Males	61(83.56%)	12(16.43%)	<0.01
3	Smokers	45(78.91%)	12(16.43%)	<0.01
4	DM	36(83.72%)	7(16.27%)	<0.01
5	SHT	41(83.67%)	8(16.32%)	<0.01
6	AWMI	58(76.31%)	18(23.68%)	<0.01
7	IWMI + I + P + RV	22(91.66%)	2(8.33%)	<0.01



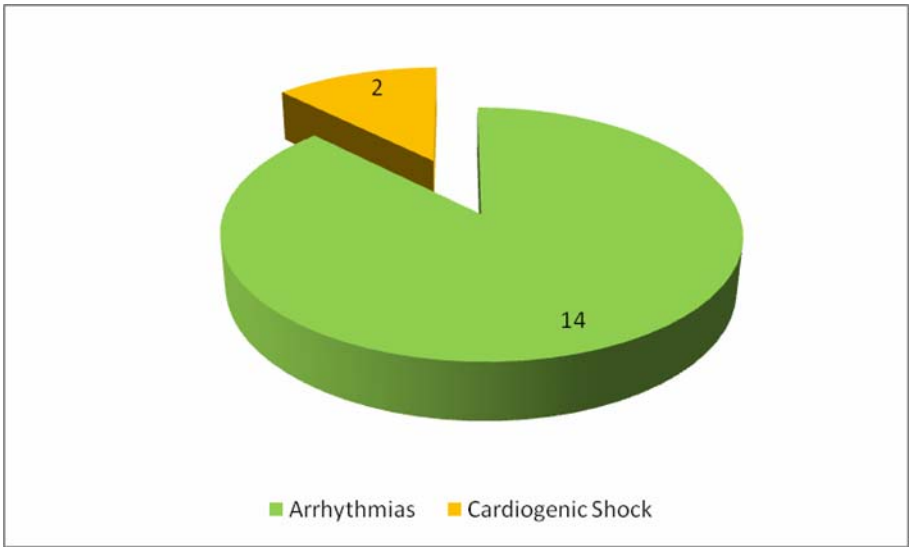
Arrhythmias are most common in males (83.56%) than females (70%)

Patients with traditional risk factors like DM, SHT, Smoking had higher incidence of arrhythmias when compared to non smokers, Euglycemics and normotensive patients.

Table 13

MORTALITY IN AMI

Total no. Of deaths	Arrhythmias	Cardiogenic Shock
16	14	2



16patients (9 males and 7 females)died out of which 2 patient were in cardiogenic shock other 14 patients had arrhythmias. 81.25% of patients had AWMI and 12.5% had IWMI.

Table 14

PERCENTAGE OF MORTALITY IN RELATION TO LOCATION OF AMI

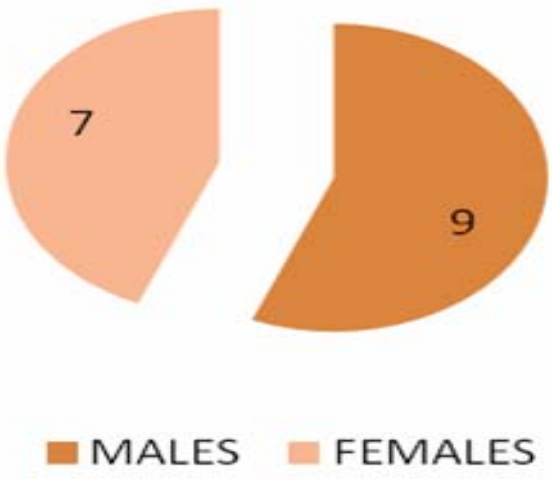
Total Mortality	AWMI		IWMI & I + P + R	
	No.Of Cases	%	No.Of Cases	%
16	13	81.25%	3	12.5%

16patients (9 males and 7 females)died out of which 2 patient were in cardiogenic shock other 14 patients had arrhythmias. 81.25% of patients has AWMI and 12.5% had IWMI.

Table 15

SEX DISTRIBUTION OF MORTALITY

SEX	DEATHS
MALES	9
FEMALES	7
TOTAL	16



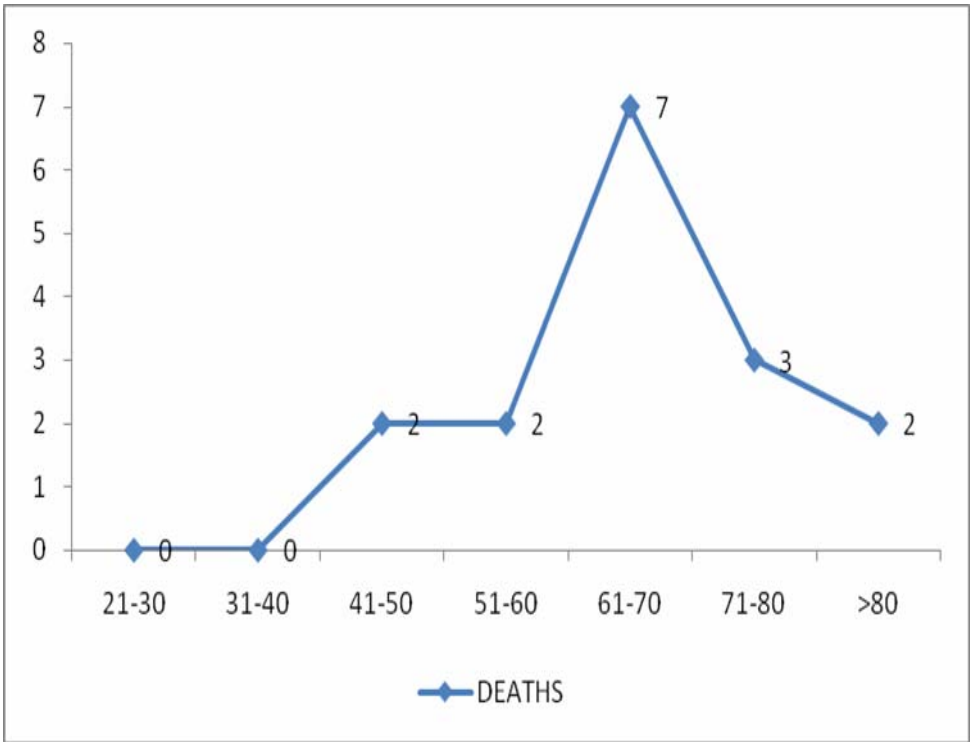
16patients died out of which 9 are male patients and 7 are female patients . The incidence of mortality is more or less equal in males and females.

Table 16

AGE DISTRIBUTION OF MORTALITY

AGE DISTRIBUTION	DEATHS
21-30	NIL
31-40	NIL
41-50	2
51-60	2
61-70	7
71-80	3
>80	2

.

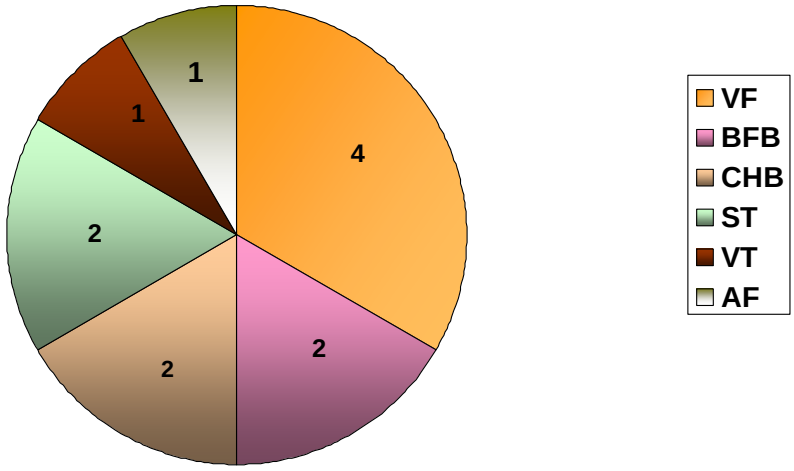


Highest mortality rate was observed in patients of 61-70 years age group

Table 17

Mortality in relation to the type of arrhythmias

Sl no	Type of arrhythmias	Deaths
01	VF	4
02	BFB	2
03	CHB	2
04	ST	2
05	VT	1
06	AF	1



Out of 16 deaths 4 patients had ventricular fibrillation , 2 patients had bifascicular block , 2 patients had complete heart block, 2 patients had

persistent sinus tachycardia, one patient had ventricular tachycardia, one patient had atrial fibrillation.

EJECTION FRACTION:

Mean EF % in patients with supraventricular arrhythmias, Bundle branch block and VT/ VF are 43+/- , 43+/- and 38+/- respectively . EF in non arrhythmic population is 48+/- .P value is < 0.05.

Patients with anterior wall MI and arrhythmias had lower EF (41%) than patients with Inferior wall MI and arrhythmias (48%).

FOLLOW UP:

30 day follow up was done only 70% of the patients came for follow up in the cardiology outpatient department .Incidence of cardiac failure (55%) was observed in patients with anterior wall MI and arrhythmia (n=58) compared to those of with inferior wall MI and arrhythmias (23%).

DISCUSSION

This study – Arrhythmias in the golden hour of myocardial infarction was conducted at Tirunelveli Medical College analyzing 100 patients with acute myocardial infarction, has elucidated many interesting observations.

DEMOGRAPHICS OF PATIENTS WITH MYOCARDIAL INFARCTION & ARRYTHMIAS

With hundred patients with acute myocardial infarction, 80% had arrhythmias whereas 20 % did not. Of 100 patients with MI, 73 %were males and 27% were females. It was seen that 90.16 % of males with MI had arrhythmias whereas only 70.37 % of females developed arrhythmias in the first hour. Highest incidence of MI was observed in the age group of 51 to 60 years in males whereas in females the maximum incidence of MI was from to 61 to 70 years. Highest incidence of arrhythmias was noted in the age group of 41 to 50 years (87.5%).

According to NCEP (National Cholestrol Education Programme),[53] (Bethesda, MD, Natonal Heart, Lung, Blood Institute, NIH,2001) decades of observational meta-analysis of various studies have clearly showed that there is higher incidence of MI and arrhythmias in males compared to Pre-menopausal women. However after menopause the coronary risk and risk of arrhythmias equals to that of men.

This is clearly demonstrated by our study as well. In the age group 31 to 40 years 60% of males with acute MI developed arrhythmias where there were no females in this age group with MI. On the other hand in the age group 61 to 70 years there were equal number of males and females with Acute MI and arrhythmias (69.23%). Hence our study conforms to previous reports.

RISK FACTOR ANALYSIS

90.16% of males and 70.37% of females had arrhythmias. Analysing single risk factors, smoking was the single most important risk factor for the development of MI(76.77%); however among the smokers with MI 80.3 % developed an arrhythmia. 43% of patients with MI were diabetic and 49 % of patients with systemic hypertension developed arrhythmias (83.7 and 83.6 % respectively). Hence diabetic and hypertensive patients were more likely to develop arrhythmias because these patients more likely to have triple vessel disease. 95 % of women were postmenopausal and had significant risk of arrhythmia.

According to a study published in NEJM, "Individual risk factors contributing to arrhythmogenesis in patients MI"- Bravier et al,[54] NEJM, 1983 – smokers had a higher incidence of MI(76%) of whom 70% developed an arrhythmia. Diabetics and hypertensives had a high incidence of MI – 69 and 73 % respectively of whom around 80%

diabetic and 69 % hypertensives developed an arrhythmia. This was in concordance to our study.

CO-RELATION BETWEEN LOCATIONS OF MI & ARRHYTHMIAS

Anterior wall MI was the most common, seen in 66% of patients. Of this extensive anterior wall MI was associated with higher incidence of arrhythmias – 85.7 %. Inferior wall MI patients had an incidence of 81.88% of which majority (40.9%) had sinus bradycardia.

Patients with anterior wall MI had more incidence of tachyarrhythmias, on the other hand patients with inferior/posterior/RV MI had more incidence of bradyarrythmias. Sinus tachycardia was the most common in anterior wall MI (31.03%) followed by Ventricular tachyarrythmias(22.41%) and AIVR(15.51 %).

Sinus bradycardia(40.9%) was the most common type seen in IWMI followed by VPB(22.7%) and 1st and 2nd degree AV lock (18%).

According to a study conducted by Hreybe H et al, Cardiology[55] Department of the Medical College of Georgia, Augusta, Georgia, USA – "Location of acute myocardial infarction and associated arrhythmias" patients with inferior wall MI were more likely to develop bradyarrythmias in contrast to patients with anterior wall MI who showed

a predilection to develop tachyarrhythmia's. This was in concordance to our study.

According to an interesting article by Sorin J. Brener, David Tschopp "Complications of Acute Myocardial Infarction" – In anterior wall MI the most common arrhythmia noted was AIVR(50%) followed by VT/VF in 4 to 8 %. Barring sinus tachycardia the higher incidence of VT/VF in our study – 22.41% can be explained by the fact that this was a small study group of only 100 patients of which majority of patients with VT/VF had multiple risk factors.

In inferior wall MI the incidence of complete heart block was 20% which was slightly higher compared to our study (13.63%). The overall trends of arrhythmias parallel each other.

MORTALITY IN RELATION TO MI AND ARRHYTHMIAS

Total mortality was 16% of which 14 patients died of cardiac arrhythmia and 2 % died of cardiogenic shock. Of the arrhythmias 4% had VF that did not respond to Cardioversion. 2 % had complete heart block and another 2 % had bifasicular block. 2 % of patients had persistent sinus tachycardia in the first hour of MI which after 6 hours degenerated into VF and could not be reverted electrically. One patient had haemodynamically unstable AF and died.

According to the result analysis of the GUSTO-III trial – "Sustained ventricular arrhythmias and mortality among patients with acute myocardial infarction", The 30 day mortality rate was 44% who experienced VT/VF died compared to our study of 33% 30 day mortality rate. This discrepancy can probably be explained by the fact that in our study only patients who were admitted in the first hour were taken up for study and early intervention was possible.

According to a study – "Survival and follow-up after pacemaker implantation after MI: patients with complete heart block -Alt E, Völker R e al – after IWMI when a patient developed complete heart block, when a pacemaker was inserted the mortality rate was low in the order of 2 to 5%, however if no intervention was done the mortality rate was high as 47.5%. According to Braunwald 5 to 10 % of patients with STEMI had more predilection for bifasicular block and higher in hospital mortality rate.

In our study 3 patients developed CHB and 2 patients died indicating a mortality rate of 66%. This was because electrophysiological intervention was not possible in our hospital setup as an emergency procedure. According to Braunwalds textbook of medicine atrial fibrillation in MI is associated with increased mortality and stroke particularly in patients with anterior wall infarction. Atrial fibrillation

itself is a rare arrhythmia in MI. In our study 2 patients had developed AF of whom 1 died.

The presence of persistent sinus tachycardia is an indirect evidence of significant LV dysfunction and predicts higher mortality rates, either patients developing persistent heart failure or degeneration into dangerous ventricular arrhythmias.

Mortality rate was more or less equal in males (56.25%) and females (43.75%).

But the incidence of arrhythmias in females was significantly less (70.37%) when compared to that of males 90.16%. Bigger et al 1978[56] and Teylor et al 1980[57] indicated that the extend of left ventricular damage is a major determinant of survival in patients following. Acute myocardial infarction, especially after anterior wall infarction.

This is concordant with our study as well that there is 81.25% mortality was observed in patients with anterior wall infarction. Gang et al (1984)[58] believed that when there was an extensive infarction, there was a greater chance for the reentry mechanism to operate, resulting in the genesis of arrhythmias.

CONCLUSION

1. Incidence of myocardial infarction increases with age.

2. Incidence of myocardial infarction is significantly more in men (73%)compared to that in women (27%).

3. In women acute MI was observed mostly (95%) in the post menopausal group.

4. Smoking was the most common risk factor for myocardial infarction (76.77%)

5. Incidence of arrhythmias are more common in diabetic and hypertensive patients (83.7%)

6. Patients with anterior wall myocardial infarction had a high risk of arrhythmic complications (76%) than patients with MI in other locations (24%).

7. Most common type of arrhythmias observed was tachyarrhythmias (80%) than bradyarrhythmias (20%).

8. Sinus tachycardia (18%) is the most frequent type observed followed by VT + VF (13%)

9. Next most common type are VPDS (12%)

10. Mortality rate was higher in older patients.

11. Most dangerous arrhythmias was VF and mortality rate is higher in patients with VF (NO=4)

Still there is higher incidence of arrhythmias and complications was observed in patients with acute myocardial infarction in resource poor settting. So early diagnosis, prompt recognition and institution of appropriate therapy (drugs, electrical cardioversion) may improve the out come.

PROFORMA

Sr. No	IP NO.
NAME	DOA
AGE	DOD
SEX	
Presenting complaints;	Smoking
Chest pain	Alcohol
Dyspnoea	Atherosclerosis
Palpitation	Family History
Syncope	Clinical Examination
Edema	General Examination
PND	Skin Xanthomas
Orthopnoea	Pulse
Vomiting	Blood Pressure
Sweating	System Examination
Polyuria	Cardiovascular system

Risk factors Respiratory system

Hypertension Gastro intestinal system

Diabetes mellitus Central Nervous system

Obesity Musculoskeletal system

INVESTIGATIONS;

Blood - Haemoglobin

Total count

Differential count

ESR

Sugar – Fasting

Postprandial

Urea

Creatinine

Lipid profile

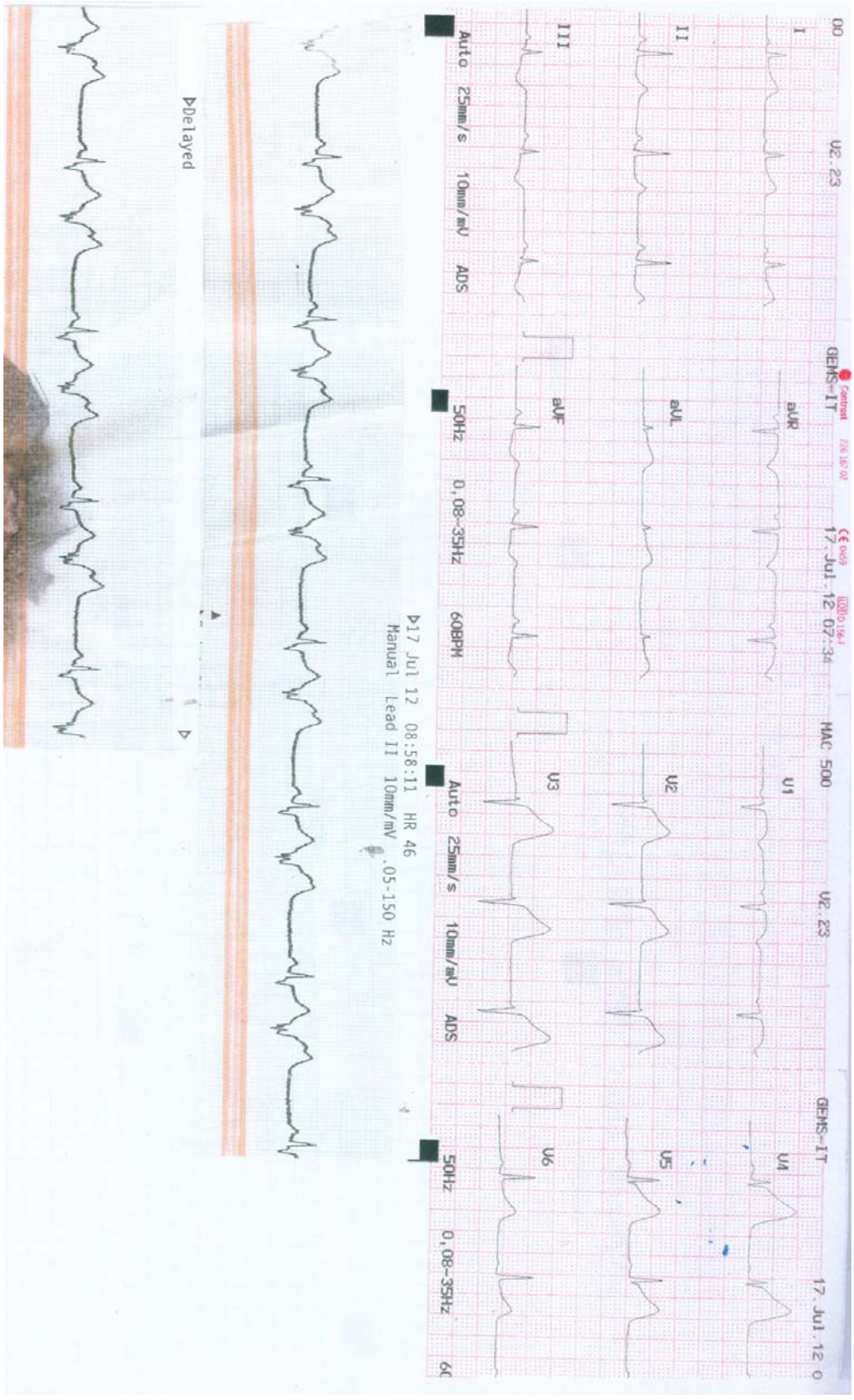
Troponin – T

ELECTROCARDIOGRAPHY

ECHOCARDIOGRAPHY

Chest X-ray PA view

ANNEXURES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GEMS-IT
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II
III
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V1
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V6
Auto 25mm/s 10mm/mV ADS
50Hz 0,08-35Hz 60BPM
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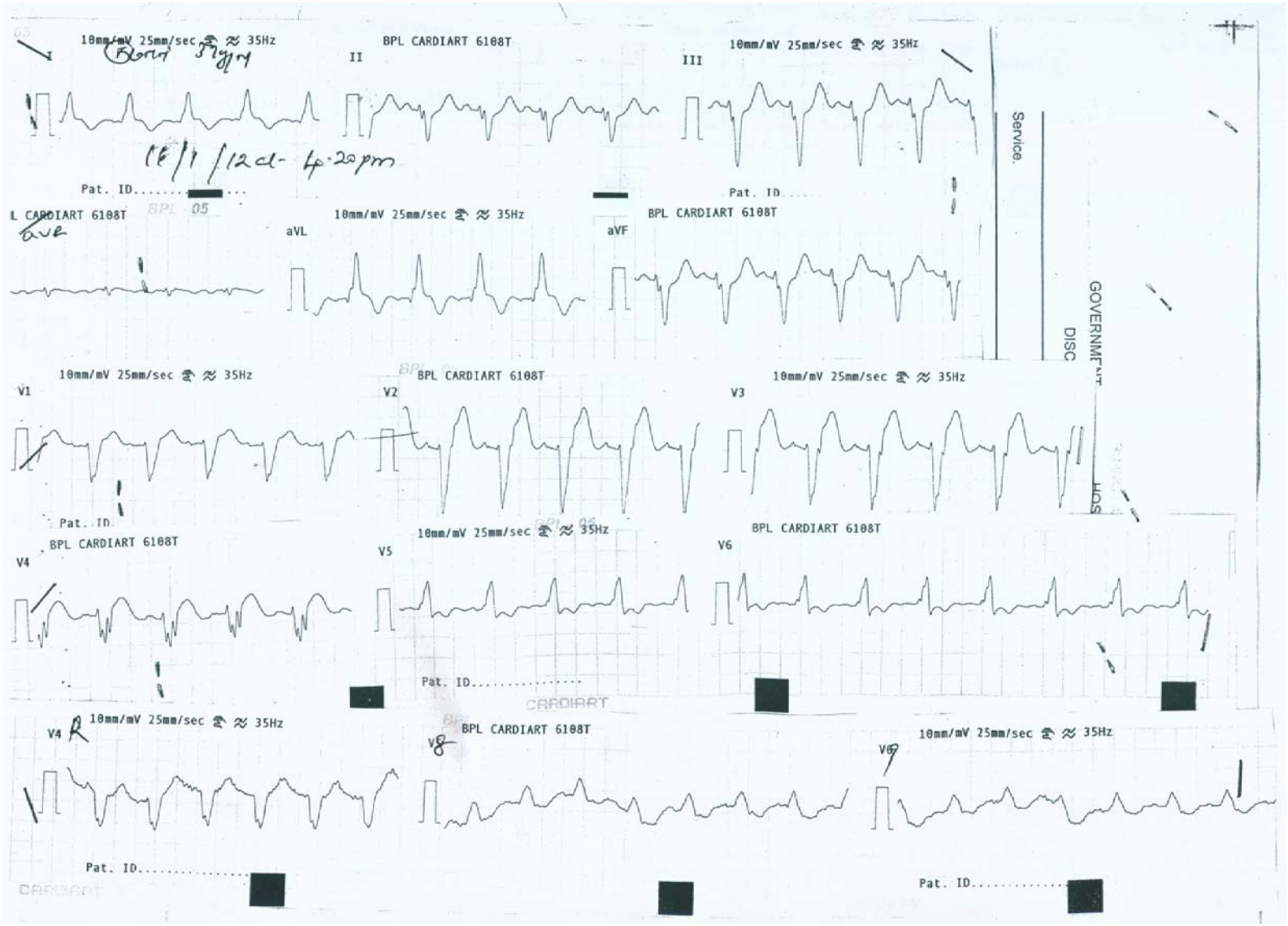
10mm/mV 25mm/sec ≈ 35Hz
BPL CARDIART 6108T
II
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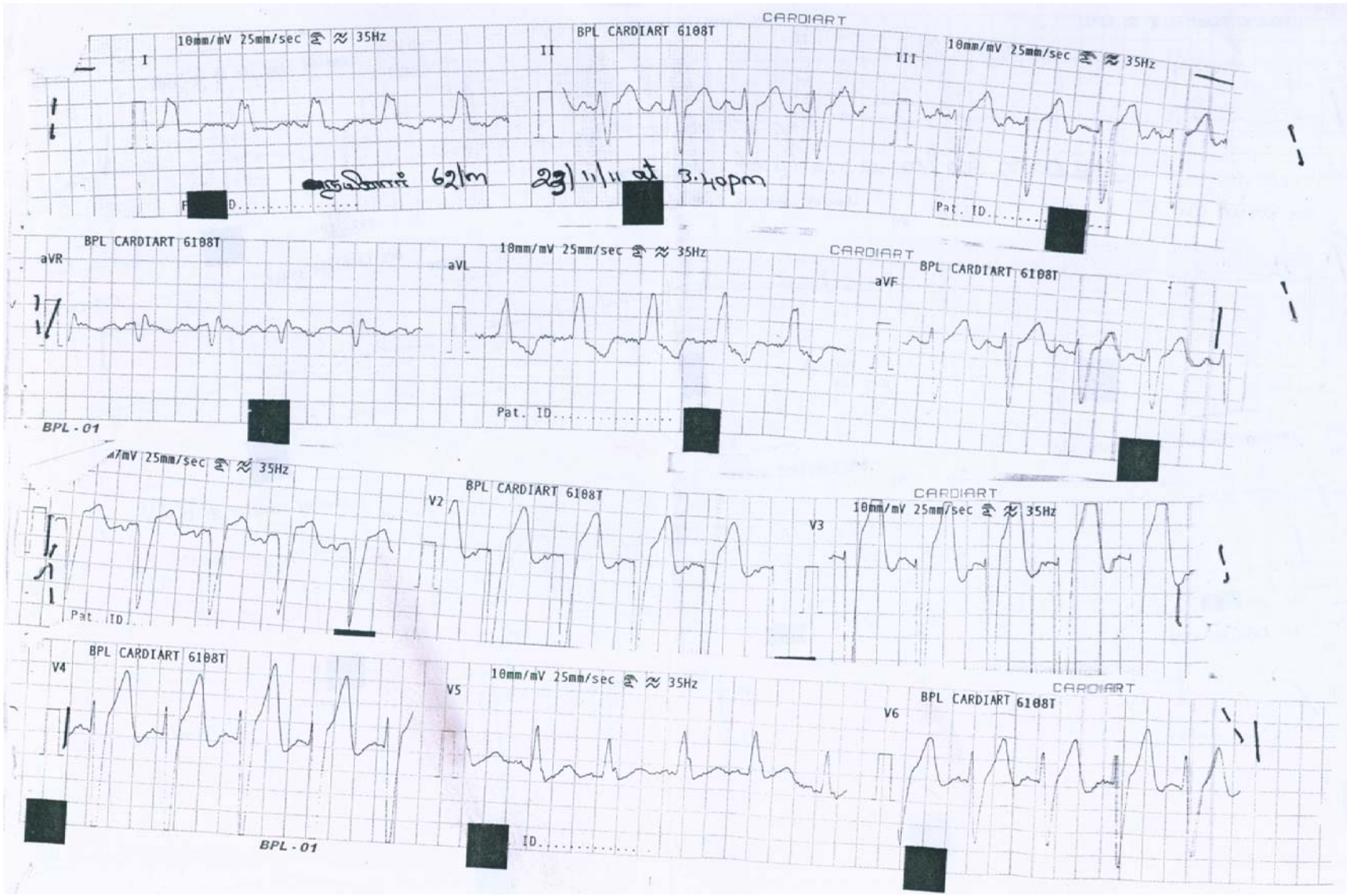
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10mm/mV 25mm/sec ≈ 35Hz
I
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BPL CARDIART 6108T
aVL
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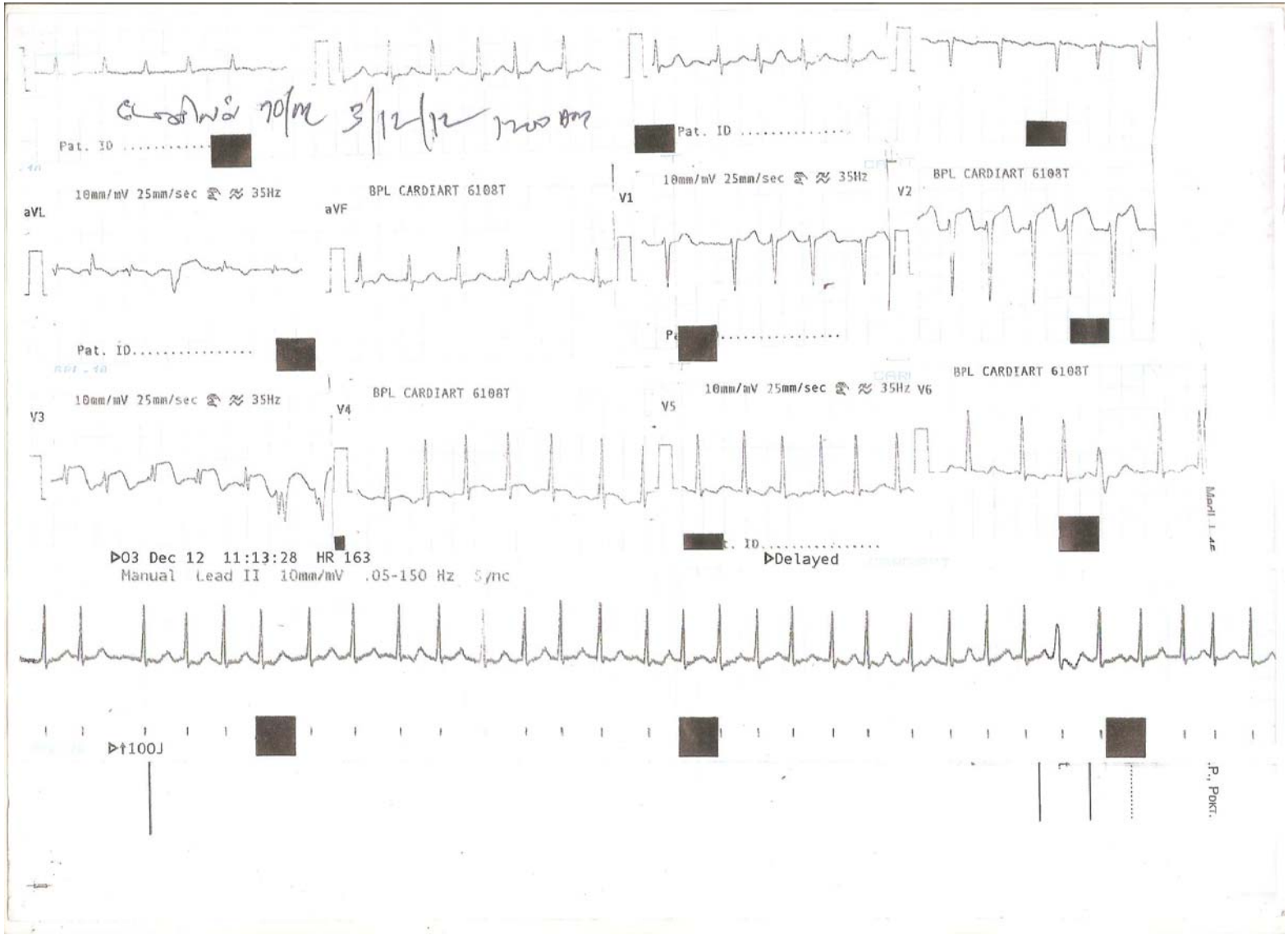
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10mm/mV 25mm/sec ≈ 35Hz
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V4
V5
V6
▷03 Dec 12 11:13:28 HR 163
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KEY TO MASTER CHART

DOA	- Day of Admission
IP. NO	- Inpatient number
DM	- Diabetes Mellitus
SHT	- Systemic Hypertension
ARR	- Arrhythmias
R	- Regular
IR	- Irregular
S	- Haemodynamically stable
US	- Haemodynamically unstable
TP	- Temporary Pacemaker
Echo +	- Echocardiogram with LV Dysfunction
ASMI	- Anteroseptal Myocardial Infarction
AWMI	- Anterior wall myocardial infarction
EAWMI	- Extensive Anterior Wall Myocardial Infarction
ALMI	- Antero lateral myocardial infarction
ST	- Sinus Tachycardia
SB	- Sinus Bradycardia
SVT	- Supraventricular Tachycardia
AF	- Atrial Fibrillation
CHB	- Complete Heart Block

LBBB - Left Bundle Branch Block

RBBB - Right Bundle Branch Block

BFB - Bi Fascicular Block

VT - Ventricular Tachycardia

VF - Ventricular Fibrillation

VPD - Ventricular Premature Depolarisation

D - Death

Sl.No	Name	Age	Sex	DOA	Time of Presentation	IP No:	SYMPTOMATOLOGY					RISK FACTORS								On		
					(HRS)		Angina	Sweating	Palpitations	Breathlessness	Syncope	Obesity	IHD	DM	SHT	Smoking	Alcoholism	Menopausal	Hypercholesterolemia	PR	Rhythm	BP
							87	36	27	33	10	33	26	52	58	59	61	26	92			
1	Sankaran	45	M	4/11/2011	1	54582	+	+	_	+	_	_	_	+	_	+	+	_	+	##	R	S
2	Munisamy	53	M	5/11/2011	1	54698	+	+	_	_	+	+	_	+	+	+	_	_	+	88	R	S
3	Mariappan	67	M	5/11/2011	1	54712	+	-	_	+	_	+	+	_	+	+	+	_	+	82	R	S
4	Karupagaraj	51	M	9/11/2011	1	54798	+	+	+	_	+	_	+	+	_	+	_	_	+	##	IR	U
5	Abdul Hameed	47	M	14-11-11	1	54867	+	+	-	_	_	_	_	_	_	_	+	_	+	78	R	S
6	Natarajan	53	M	14-11-11	1	54879	-	-	_	_	_	_	_	+	_	+	+	_	+	82	IR	S
7	Janaki	60	F	17-11-11	1	54962	-	+	_	+	+	+	_	+	_	_	_	+	+	48	R	S
8	Sulochana	68	F	26-11-11	1	54997	+	-	_	+	_	+	_	_	+	_	_	+	+	82	R	S
9	Manoharan	55	M	29-11-11	1	55002	+	-	+	+	_	_	_	_	+	+	+	_	+	114	R	S
10	Mohan Kumar	42	M	3/12/2011	1	55058	-	-	_	_	_	_	_	_	_	+	_	_	+	70	IR	S
11	Manga	63	F	7/12/2011	1	55132	+	+	+	+	+	+	+	+	+	_	_	+	_	##	R	U
12	Arunachalam	72	M	7/12/2011	1	55167	+	-	_	_	+	_	_	_	_	+	_	_	_	36	R	S
13	Loganathan	57	M	16-12-11	1	55413	+	+	_	_	_	_	_	_	+	+	+	_	+	90	R	S
14	Petciyammal	70	F	18-12-11	1	55497	-	+	_	+	_	+	_	+	+	_	_	+	+	84	R	S
15	Palpandi	48	M	21-12-11	1	55582	+	+	+	_	+	_	_	_	_	+	+	_	+	##	R	S
16	Govindaraj	43	M	28-12-11	1	55712	+	-	_	_	_	_	_	_	+	+	_	_	+	96	R	S
17	Nasariah	60	M	1/1/2012	1	55819	+	-	_	_	_	_	_	+	_	+	_	_	+	92	IR	S
18	Kayanchi	56	M	7/1/2012	1	55904	-	+	+	+	_	_	_	_	+	+	_	_	+	112	R	S
19	Karpagaraj	63	M	15-1-12	1	56016	+	-	+	_	+	_	+	+	_	+	_	_	+	##	R	U
20	Mani	53	M	15-1-12	1	56031	+	+	_	_	_	+	_	_	+	+	+	_	+	88	R	S
21	Radhakrishnan	80	M	19-1-12	1	56117	+	-	+	_	+	_	+	+	_	+	+	_	+	92	IR	V
22	Saraswathiammal	61	F	23-1-12	1	56195	-	+	_	+	_	+	_	+	_	_	_	+	+	74	R	S
23	Chandrasekhar	52	M	26-1-12	1	56256	+	-	+	_	_	+	_	_	+	+	+	_	+	##	R	S
24	Ponnusamy	64	M	30-1-12	1	56302	+	-	_	_	_	_	_	+	_	_	_	_	_	90	R	V
25	Ramasubramaniyam	57	M	8-2-12-	1	56424	+	-	_	+	_	+	_	_	_	+	+	_	+	87	R	S
26	Velayudham	56	M	13-2-12	1	56553	+	-	_	_	_	_	_	_	_	+	+	_	+	52	R	S
27	Marimuthu	82	M	22-2-12	1	56712	+	+	_	+	_	_	_	+	+	_	+	_	+	##	R	U
28	Thavammal	75	F	26-2-12	1	56807	+	+	_	_	+	+	_	_	+	_	_	+	+	68	R	S
29	Selvan	66	M	1/3/2012	1	56893	+	+	+	_	_	_	_	+	+	+	+	_	+	##	R	S
30	Durairaj	59	M	5/3/2012	1	56992	_	+	+	_	_	_	_	+	_	+	_	_	+	113	R	S

31	Karuppiah	58	M	5/3/2012	1	56997	+	-	_	_	_	_	_	_	+	+	_	_	+	84	IR	S	
32	Abdul wakab	60	M	11/3/2012	1	57051	+	-	_	+	_	+	+	_	+	+	_	_	+	88	R	S	
33	Susheela	60	F	18-3-12	1	57102	+	-	_	_	_	+	_	+	_	_	_	+	_	82	R	S	
34	Shankar	62	M	22-3-12	1	57159	+	-	_	_	+	_	_	_	+	+	_	_	_	78	IR	S	
35	Nagarathinam	61	F	27-3-12	1	57218	+	-	_	+	_	+	_	_	+	_	_	+	+	92	R	S	
36	Dileep	28	M	31-3-12	1	57287	+	-	+	_	+	_	_	_	_	+	+	_	_	118	R	S	
37	Raghupathy	70	M	4/4/2012	1	57332	+	-	_	+	_	_	_	+	+	_	_	_	+	92	IR	S	
38	Packiyanathan	44	M	6/4/2012	1	57423	+	+	+	_	_	_	_	+	_	_	+	_	+	114	R	S	
39	Ponnusamy	57	M	13-4-12	1	57581	-	+	_	+	_	_	_	+	_	+	_	_	+	66	R	S	
40	Muthumari	45	F	15-4-12	1	57669	+	+	_	_	_	+	_	_	+	_	_	_	+	86	R	S	
41	Ellammal	65	F	16-4-12	1	57694	+	+	+	_	+	+	+	_	+	_	_	+	+	##	R	U	
42	Muniyammal	60	F	23-4-12	1	57762	+	-	_	+	+	_	+	+	_	_	_	+	+	68	IR	S	
43	Basha	78	M	28-4-12	1	57887	+	-	+	_	_	_	+	_	+	+	_	_	+	##	R	S	
44	Petchithai	80	F	29-4-12	1	57902	+	+	_	_	+	+	_	_	+	_	_	+	+	38	R	S	
45	Abraham	38	M	2/5/2012	1	57964	+	-	_	_	_	_	_	+	_	+	_	_	+	78	IR	S	
46	Nandakumar	40	M	6/5/2012	1	57997	+	-	_	_	_	_	_	_	_	+	_	_		78	R	S	
47	Sundar	44	M	#####	1	58132	+	-	_	_	_	_	_	_	+	+	_	_	+	74	R	S	
48	Chinnasamy	49	M	16-5-12	1	58213	+	-	_	+	_	_	_	_	_	_	_	_	+	94	IR	S	
49	Muthukumar	55	M	21-5-12	1	58284	+	+	+	_	_	_	_	_	+	+	_	_	+	##	R	S	
50	Arjunan	73	M	27-5-12	1	58321	+	+	_	_	_	+	+	+	+	+	_	_	+	76	R	S	
51	Mohammed	77	M	31-5-12	1	58384	+	-	_	_	_	_	_	_	+	+	_	_	+	46	R	S	
52	Chandran	58	M	1/6/2012	1	58392	+	-	_	+	_	_	_	+	+	+	_	_	_	78	R	S	
53	Mohan kumar	52	M	7/6/2012	1	58462	-	+	+	+	_	_	_	+	_	+	+	_	_	84	R	S	
54	Shanthi	50	F	#####	1	58514	+	-	+	_	_	+	_	_	_	_	_	+	+	78	R	S	
55	Umadevi	78	F	14-6-12	1	58596	_	+	_	+	+	_	_	+	+	_	_	+	_	64	IR	S	
56	Narayanasamy	44	M	21-6-12	1	58693	+	-	+	_	_	_	_	_	_	_	_	_	+	86	IR	S	
57	Soundararajan	47	M	27-6-12	1	58772	+	-	+	_	_	_	_	_	_	+	_	_	+	110	R	S	
58	Mary	61	F	3/7/2012	1	58863	+	-	+	_	_	+	_	_	+	_	_	+	_	98	IR	S	
59	Dhanapal	29	M	6/7/2012	1	58918	+	+	-	+	+	_	_	_	_	+	_	_	_	52	R	S	
60	Arivuselvam	56	M	18-7-12	1	59102	+	-	-	+	+	_	_	_	_	+	_	_	+	88	R	U	
61	Pooammal	65	F	20-7-12	1	59144	+	-	-	+		+	+	+	+		_	+	+	82	R	S	
62	Ravi	53	M	25-7-12	1	59247	+	-	+	+	+	_	_	_	_	+	+	_	_	##	R	U	
63	Ramalakshmi	60	F	28-7-12	1	59304	+	-	-	+	+	_	_	+	_	_	_	+	+	56	R	S	

64	Rukmaniammal	63	F	30-7-12	1	59391	+	+	-	_	_	+	+	+	_	_	_	+	+	76	R	S	
65	Ezhumalai	52	M	2/8/2012	1	59424	+	-	-	_	+	_	_	_	_	_	_	_	+	48	R	S	
66	Ramu	48	M	5/8/2012	1	59499	+	+	+	+	+	_	_	_	+	+	+	_	+	##	R	U	
67	Ramamurthy	57	M	11/8/2012	1	59592	+	-	-	+	+	_	_	+	+	_	_	_	+	62	IR	S	
68	Yuvaraj	42	M	17-8-12	1	59680	+	-	+		+	_	_	_	_	+	+	_	+	##	R	S	
69	Santhosh	49	M	23-8-12	1	59726	+	-	_	+	_	_	_	+	+	+	_	_	+	78	R	S	
70	Munisamy	39	M	31-8-12	1	59831	+	+	_	_	_		+	+	_	_	_	_	+	67	IR	S	
71	Syed Ali	74	M	4/9/2012	1	59927	+	-	_	_	_	+	+	_	+	+	_	_	+	88	R	S	
72	Sudalaimuthu	70	M	9/9/2012	1	59993	+	-	_	_	_	_	+	+	+	+	_	_	+	114	R	S	
73	Manickam	55	M	11/9/2012	1	60013	+	+	+	_	_	_	_	_	+	+	_	_	V	116	R	S	
74	Pushpammal	52	F	#####	1	60089	-	+	_	+	_	+	_	+	_	_	_	+	_	52	R	S	
75	Natarajan	67	M	15-9-12	1	60108	+	+	+	_	_	_	+	+	+	+	_		+	##	R	U	
76	Mahalingam	64	M	17-9-12	1	60175	+	-	_	_	_	_	_	_	_	+	_	_	+	84	R	S	
77	Ramachandran	56	M	23-9-12	1	60286	+	-	_	_	_	_	_	+	_	_	_	_	+	78	R	S	
78	Malliga	52	F	27-9-12	1	60377	-	-	_	+	_	+	_	_	+	_	_	+	+	##	R	S	
79	Karunakaran	50	M	30-9-12	1	60465	+	-	+	_	+	_	_	+	+	_	_	_	+	74	IR	S	
80	Selvarani	43	F	30-9-12	1	60473	+	+	_	_	_	+	_	_	_	_	_	_	+	86	R	S	
81	Saradha	73	F	1/10/2012	1	60511	+	-	_	+	_	+	_	+	+	_	_	+	+	5	R	S	
82	Rajaram	45	M	#####	1	60572	+	+	+	_	_	_	_	_	+	+	_	_	_	88	IR	S	
83	Thangaraj	37	M	#####	1	60703	+	+	_	_	_	_	_	_	_	+	+	_	+	88	R	S	
84	Shahul Hameed	66	M	#####	1	60717	+	+	+	+	_	_	_	+	_	+	_	_	+	64	IR	S	
85	Purushothaman	56	M	#####	1	60802	+	+	+	_	+	_	+	_	+	+	_	_	+	##	R	U	
86	Ummar	57	M	#####	1	60879	-	+	+	+	_	+	+	+	+	+	+	_	+	##	R	S	
87	Lekshmi	70	F	#####	1	60896	+	-	_	_	+	_	_	+	_	_	_	+		92	IR	U	
88	Rajeswari	64	F	19-10-12	1	61003	+	-	_	_	_	+	_	_	+	_	_	+	+	##	R	S	
89	Deivasigamani	65	M	24-10-12	1	61076	+	+	_	_	_	+	+	+	_	+	_	_	+	76	R	S	
90	Elizabeth	70	F	25-10-12	1	61087	+	+	+	_	_	_	+	_	+	_	_	+	_	##	IR	U	
91	Krishnan	71	M	28-10-12	1	61102	+	+	_	+	_	_	+	_	+	+	+	_	+	94	R	S	
92	Vellathai	60	F	1/11/2012	1	61159	+	+	_	_	_	_	_	+	_	_	_	+	+	88	R	S	
93	Govindammal	85	F	3/11/2012	1	61194	+	+	_	_	+	+	+	_	+	_	_	+	+	38	R	U	
94	Venkatiah	69	M	3/11/2012	1	61229	+	+	+	+	+	_	+	_	+	_	+	_	+	##	R	U	
95	Narayanasamy	70	M	5/11/2012	1	61284	+	-	_	_	_	+	_	+	_	+	+	_	+	92	IR	S	
96	Kalyanasundaram	53	M	6/11/2012	1	61303	-	+	+	+	_	_	_	+	_	+	_	_	_	##	R	S	

97	Tharamani	53	M	8/11/2012	1	61338	+	-	_	_	_	_	_	_	+	+	_	_	+	87	IR	S	
98	Venkatesan	44	M	#####	1	61382	+	-	+	_	_	+	+	_	_	+	+	_	+	92	R	S	
99	Joseph	38	M	#####	1	61396	+	-	+	_	_	_	_	_	_	+	_	_	_	112	IR	S	
100	Muthukrishnan	34	M	#####	1	61427	+	+	+	_	+	_	_	_	_	+	+	_	_	94	R	S	